The College of Physicians
and Surgeons.

With the Compliments of
the Author.
Chas. T. Parkhurst
N. S. 1890.

THE
INTESTINAL PUTREFACTIONS

CLINICAL STUDIES

OF

ENTEROCOLITIS

BY

CHARLES FENNER PECKHAM, M. D.

PROVIDENCE, RHODE ISLAND

SNOW & FARNHAM CO., PRINTERS
1916

CONTENTS

CHAPTER XIII.

CHAPTER XIV.

CHAPTER XV.

PREFACE

In the year 1906, Professor Herter published his epoch-making work on the Common Bacterial Infections of the Digestive Tract. Before this, other medical thinkers had pointed out the way; Schmidt and Strasburger had published their monumental work on the human feces and Metchnikoff had noted the connection between intestinal putrefaction and the duration of human life. It remained for Herter, however, to give the impulse that led to the application of these discoveries in clinical practice. It was through his classic teaching that physicians learned to use the three great weapons: chemistry, bacteriology and biology, in the fight. Various phenomena such as hyper- and hypochlorhydria, indicanuria and autointoxication, so called, fitted themselves into the clinical pictures; diseases that at first sight seemed to be intimately connected with faulty metabolism in the human tissues were proven to arise not through any parenteral biochemical process but in spite of it. Organic lesions were given the prominent position that they deserve, while the various types of intestinal putrefaction were used as avenues of information through which a knowledge of the nature and mode of attack of the enemy might be gained.

In the preparation of this book, that great enemy of the arts, fleeting time, has, of necessity, placed certain limitations upon the task. Its preparation has been a labor of great satisfaction. By far the greatest satisfaction, however, rests in the fact that what was formerly a matter of conjecture, can through laboratory methods be brought to a point of

absolute certainty. This should give the physician, through the directness of his therapeutic attack, greater power in the battle with disease, and should he, through the methods set forth in the following pages, win the fight, prolong the useful life or even alleviate the suffering of some unfortunate human being, the writer will feel that his labors have been well repaid.

The Author.

Six Thomas Street,
Providence.

CHAPTER I

ENTEROCOLITIS

The course of this disease may be divided into three stages, and these for convenience in clinical study may be termed primary, secondary and tertiary. In the primary stage a sudden onset accompanied by more or less acute symptoms is the rule. The secondary stage is marked by a period of increasing gastrointestinal disturbance, either continuous or intermittent, extending over a considerable length of time. The tertiary stage begins with the advent of low-grade inflammatory changes involving the stroma and parenchyma of some organ or group of organs of the body.

The duration of the primary stage is a matter of days, that of the secondary stage may be reckoned in years or decades, while that of the tertiary stage depends upon the vitality of the organs involved. The primary stage may begin in early infancy, in childhood, in adolescence or at any time during the life of the patient. Many of the obscure febrile disturbances of infancy and childhood, while not recognized at the time as the beginning of a disease that may lead to inefficiency and premature old age, mark the beginning of a chronic enterocolitis.

Certain families seem especially liable to the ravages of this disease and upon questioning these patients, it will be found that their ancestors for many generations have died of diseases traceable to intestinal putrefaction, such as arteriosclerosis, cirrhosis of the liver and other low-grade inflammatory disturbances in various specialized organs, at ages well below sixty.

Errors in diet, constipation, overindulgence in animal food are not of such importance in the etiology of enterocolitis

as is the reduction of vitality incident to the stress and competition of modern life and the lack of nourishing food and fresh air.

As the most prominent predisposing cause, the habitual use of purgatives and cathartics, especially the salines, may be given a prominent position. The irritation of the intestines, through the daily use of sodium phosphate, sodium sulphate, magnesium sulphate and potassium bitartrate so lowers the resistance of the intestinal mucous membrane as to encourage the invasion of the infecting micro-organisms. Cathartics of less irritating character may also be a very prominent factor in the development of this condition.

To the results of emotional excitement, grief, disappointment and other depressants of the resisting power, may be added the exposure to strains of bacteria with which the patients were not accustomed to deal.

Before beginning the treatment of these patients, three diagnoses should be made:

First: The anatomical diagnosis, dealing with the nature and extent of the alimentary catarrhal process, the position of the abdominal viscera and the condition of the liver, heart, kidneys and other internal organs.

Second: A chemical diagnosis directed to estimating the patient's capacity for oxidizing acids and toxins and to determine the nature of the intoxication.

Third: A biological diagnosis covering the character of the intestinal fauna, and the presence of parasites in the tissues of the body.

Primary Stage.

In the primary stage the clinical picture is one of acute infection. There may be a rise in temperature preceded by a chill, anorexia and vomiting. The local symptoms may not be pronounced but pains in various portions of the abdomen are prominent if the colon is markedly involved. These pains are generally referred to some of the flexures

of the large intestine. If, however, parts of the alimentary canal higher up are affected the pain is referred to the epigastrium. Often mucous stools, stained saffron yellow, will be noted. This disturbance may vary in severity from one so slight as to pass unnoticed to toxemia so overwhelming as to cause sudden death. Other subsequent attacks do not generally equal the first in severity. There may be constipation or diarrhoea.

Secondary Stage.

Convalescence from the primary stage of this disease may appear to the patient to be complete. A chemical or microscopical examination of the patient's feces, urine and blood, however, will show that a marked change has taken place. That a condition has developed that if allowed to go on, will eventually impair the usefulness of the victim, or lead to a premature decay of some or all of the functions of his body. Unlike the primary stage, this is of long duration, running a course of years or even decades. It is a stage when changes generally take place gradually, when like the mills of the gods the grinding process is slow but the grist exceeding fine. Ten, twenty or even thirty years may pass before a vital organ begins to suffer changes, but these changes are as sure to come as the changes of the seasons.

This stage is characterized by dyspeptic symptoms, described by the patient as severe or slight, as the susceptibility or resistance of the individual may vary. These complaints may be transitory or continuous.

It is unfortunate that the gastro-enteric mucosa is not more liberally endowed with the sense of pain and that very few intestinal lesions give this symptom in a marked degree until the disease has progressed far enough to involve the muscular or peritoneal coat. The intestinal mucosa often suffers such damage that the nutrition of the individual is irreparably undermined, with no other alarming symptom, perhaps, than a more rapid loss of flesh than a dyspepsia might cause.

The symptoms and physical signs of this stage may be classified as general and local. The general symptoms arise from the attack of toxins upon the patient's tissues, causing irritation and depression of vitality in various organs, or groups of organs. Bearing this in mind, the various clinical pictures met with in this stage, can be easily composed and their great variety fully explained.

From the patient's standpoint the course of the disease is not continuous. At the time of the acute attacks he is in misery: during the periods of remission, he enjoys comparative comfort. Each attack, however, leaves a certain amount of damage behind it, varying in amount with the natural resistance of the affected organ.

Locally, a train of symptoms and physical signs presents itself that has its origin in the direct irritation of the intestinal mucosa. Spasms, which may reach the point of producing intense pain, referred to the pylorus, the stomach or the intestines are common. The pylorus is the most frequent site of this disturbance, apparently arising from the effort of the intestine to protect itself from the irritation of the acid stomach contents. This may become so persistent as to lead to continuous nausea or prolonged vomiting. Spasms of the colon are next most frequently encountered, and often lead to spastic constipation with triangular, ribbon-like or scybalous stools of small calibre, and considerable pain and tenesmus.

While the symptoms in this Secondary Stage may be so mild as to escape ordinary notice, yet a patient in this condition of comparative comfort, may be in even greater danger than one showing the most pronounced symptoms. How often have we noted a change in physical condition gradually develop in certain people. They do not consider themselves ill, they manage to perform their daily tasks with satisfaction to themselves and to their employer, and complain, if they complain at all, only of a slight lack of their former endurance; yet they do not appear to be in perfect health. To the practiced eye, their illness may be diagnosed offhand as

progressive anemia of obscure origin, and eventually they die suddenly of cardiac, renal or hepatic diseases which are ascribed, perhaps, to overwork. The long-standing intestinal putrefaction, which was the direct cause of their untimely end, is usually overlooked.

Tertiary Stage.

When organic lesions of any organ can be demonstrated clinically, the Tertiary Stage may be said to begin. For some time before this, however, organic changes, though not apparent upon physical examination, have been slowly advancing.

The onset of this stage may be either gradual or extremely sudden. In the latter case, a diagnosis of acute inflammation is often erroneously made. If of sudden onset, the symptoms may be very violent and often so severe as to cause death. The fact should be borne in mind, however, that the lesions in the kidney, heart, blood vessels or other specialized organs have not arisen de novo, but as the result of a long-standing toxemia.

If the onset of this stage is gradual very few symptoms will at first be given. More often, however, the presence of some physical signs discovered in the course of the examination of the urine, feces or blood, or in the physical examination of the patient will disclose the fact that the fight is lost. In other individuals the sudden cessation of improvement or a rapid loss of strength and endurance show only too plainly the beginning of some organic changes.

In the case of individual organs, the importance of these to life may have a great bearing upon the subsequent course of the disease. Lesions is such organs as are intimately connected with digestion, metabolism and excretion naturally have a most deleterious effect upon intestinal catarrhs and putrefactions. Failure of the organs of circulation may bring on passive congestions or anemias that preclude the possibility of any improvement in intestinal conditions. The function of the nervous mechanism of the viscera may

be so damaged as to upset the balance of the various abdominal organs and to impair their vitality.

Death may result from the failure of some organ necessary to life or the result of some intercurrent disease to which the patient's weakened condition opens the way.

CHAPTER II

THE MICRO-ORGANISMS OF THE INTESTINAL CANAL

The micro-organisms that are the causative agents in chronic catarrhal processes affecting the gastro-intestinal canal and the various passages connecting therewith, offer a field for study, that up to the present time, has been very lightly cultivated. Limited as is the field in most cases to the study of the feces, the fauna of the higher parts of the intestinal lumen, in the human subject at least, is extremely difficult to investigate. As these diseases are rarely, per se, fatal, the patient's death being the result of the secondary changes taking place in some distant organ, we must of necessity rely upon specimens taken at the time of operation upon the upper intestinal canal or upon the gall ducts for our information. Deductions from the chemical findings in both feces and urine have been found of great assistance in throwing light upon the character of the infective process. For example, the presence of a very strong reaction to Ehrlich's aldehyde in the feces leads us to suspect a luxuriant growth of the short, plump bacillus, which in pure culture gives a remarkably sharp response to this test, in the duodenum and jejunum, even if these bacilli have disappeared by the time the fecal specimen reaches the laboratory for examination.

From the clinical standpoint the vegetable parasites of the intestinal canal and the passages directly connected with it, may be divided into three classes:

1. Those which, gaining access to the intestine, invade the tissues of the host.

2. Those which feed upon the epithelium of the mucus membrane of the intestine and rarely penetrate more deeply into the tissues.

3. Those which grow in the contents and the mucus covering the surface of the intestinal canal and do not directly attack the mucous membrane.

The group of intestinal infections herein described belong to parasitism of the third class and include the bacteria, hyphomycetes and saccharomycetes.

The effect of the growth of micro-organisms may be classified as follows:

1. The direct parasitic effect: diversion of food supply, chemical changes in the chyle, rendering it unfit for absorption.

2. The production of toxic by-products, such as acids and acid salts, aromatic bodies, protein bodies, ethereal sulphates, ammonia.

3. The effect upon the organs directly concerned in internal metabolism, including overwork in oxidizing acids, ethereal sulphates, etc., and the direct toxic effect, leading to structural changes in distant organs.

Changes in these organs may be classified as follows:

In the parenchyma: hyperplastic and increased function, atrophic and diminished function.

In the stroma: hypertrophy, fibrosis, degeneration, contraction, cirrhosis, calcification.

These changes may involve cardiac, arterial, hepatic, renal, gastro-intestinal, nervous, muscular, articular, ovarian, pancreatic or any of the various forms of animal tissue, with a syndrome corresponding to the special type of degeneration.

The animal parasites of the intestinal canal are without the matter under discussion and will only be mentioned when their presence has some bearing upon the subject at hand.

As the putrefactive processes of the alimentary canal are studied clinically and in the laboratory, the questions often arise, Why do the contents of the normal human intestine remain so nearly sterile? Why, in normal stools, do we find so few micro-organisms of the putrefactive type and such a luxuriant growth of the bacillus communis coli?

We find also that feces kept in an incubator for days do not undergo a great change in their bacterial fauna; that outside of an increase in the number of the special types present when that stool was passed, even after the specimen has been exposed to contamination in the laboratory, very few new types of micro-organisms develop.

The feces of infants may be kept in a temperature of 98.6°F. for a week or more without change and even the addition of such feces to culture media will check the growth of certain micro-organisms.*

There seems to be some antiseptic present in normal feces that has a specific or selective action against the "wild" strains of bacteria. This body is probably secreted by the liver and pancreas and by the intestinal mucosa and in health keeps all bacteria in check, except the colon bacillus peculiar to the genus homo. The strength or power of this antiseptic varies inversely with the age of the individual and by gauging its strength an accurate estimate of the relative age can be made.

An intestinal catarrh gives very little trouble until this antiseptic begins to be diminished in amount or in strength, and in no case can recovery be considered complete until repeated examinations of the feces show that this function has been regained to an extent commensurate with the age of the individual. If the antiseptic body could be produced synthetically or by extraction, it could be truly said that one of the springs of life had at last been found.

Putrefaction is never present in the intestinal canals of normal animals and when found is always an evidence of disease. When present, we may assume an impairment of the antisepticus intestinalis in the chyle and feces. This antiseptic, as we said before, is selective for every type of bacteria with which the organism habitually comes in contact, except the colon bacillus.

There are several conditions that may weaken or depress the secretion of this antiseptic—

*Herter, Com. Bac. Inf. Digestive Tract, p. 13.

(a) The general reduction of vitality through grief, pain and misfortune.

(b) The attack of toxins, such as those produced in the growth of B. tuberculosis, treponema pallida, plasmodium malariae and other micro-organisms.

(c) The direct effect of catarrhal processes upon the mucosa.

To thoroughly comprehend the process, the intestinal canal must be regarded as a hollow organ continuous with other external epithelial surfaces of the body. The contents of the intestine are, in consequence, as much outside the body as the water in which we wash our hands. There is a marked difference, however, between the mucous membrane and the skin and this is in the rate of absorption. While this for many chemical bodies is fairly active on cutaneous surfaces, on the intestinal surface it is of extreme activity. This fact is taken advantage of by the bacteria of intestinal putrefaction, which by their toxins and aggressins still further reduce the vitality of the body tissues.

The vegetable parasites may be considered under the following heads: (1) Bacteria, (2) Hyphomycetes or molds, (3) Saccharomycetes or yeasts. While this classification may not be strictly and scientifically accurate, still it answers all the requirements of clinical study.

The following list of bacteria, according to the classification of Chester will be found of great service in the study of this subject in the laboratory. This list gives most of the bacteria encountered in this class of work and for convenience of reference the number given in Chester's valuable work is prefixed.

1. BACTERIA:

A. Streptococcus:

2. Str. enteritis Hirsch.
7. Str. enteritidis Escherich.
10. Str. canis.

11. Str. coli.
15. Str. lactis.

B. Micrococus:

6. M. pyogenes albus.
20. M. aerogenes Miller.
22. M. alvi.
27. M. Mendozae.
34. M. cumulatus v. Besser.
35. M. salivarius Biondi.
43. M. ovalis Escherich.
44. M. lactis.
45. M. candicans.
61. M. subflavus v. Besser.
64. M. pyogenes aureus.

C. Sarcina:

1. Sarc. pulmonum.
4. Sarc. flava.
5. Sarc. lactis.
7. Sarc. lutescens Stubenrath.
9. Sarc. lutea.
11. Sarc. cervina Stubenrath.
12. Sarc. fusca Gruber.

D. Planococcus:

E. Planosarcina:

3. P. Sarc. Samesii.

F. Bacterium:

10. Bact. aceti.
17. Bact. acetigenum.
18. Bact. aceticum.
20. Bact. aerogenes.
21. Bact. capsulatum.
57. Bact. Bienstockii.
62. Bact. dysenteriae.
71. Bact. lactis.
166. Bact. Welchii.

166. Bact. aerogenes capsulatus.
171. Bact. thermophilum.
210. Bact. subtiliforme.
211. Bact. simile.
Bact. Mellanby & Twort.*

G. Bacillus:

2. B. coli.
5. B. enteritidis.
21. B. typhosus.
33. B. alcaligenes.
37. B. Friedbergensis.
39. B. Schafferi.
65. B. Shigae.
75. B. bucalis.
84. B. tachyctonus.
113. B. dysenteriae. Kruse.
116. B. Wesenbergii.
142. B. prodigiosus.
155. B. Lesagei.
194. B. subtilis.
238. B. putrificus.
243. B. caris.
245. B. solidus.
250. B. botulinus.
251. B. butyricus Botkin.
252. B. amylobacter—Clostridium butyricum.
254. B. Kedrowskii.
256. B. sporogenes.
B. bifidus (vide Herter p. 41).
B. acidophilus.
B. acidolacticus.
B. anthracis symptomatici.
B. cloacae.
B. entericus.
B. liquefaciens ilei.

*Protein Split Products, Vaughn, 1913, p. 294.
Journal Physiol. 1912, XLV, 53.

B. mascerans.
B. violarius acetonicus.
B. oedematis maligni.
B. paraputrificus.
B. proteus vulgaris.

H. Pseudomonas:

2. Ps. monadiformis.
29. Ps. capsulata.

I. Microspira:

6. Msp. comma.
10. Msp. protea.

J. Spirillum:

2. Sp. Massauah.

K. Spirochaeta:

L. Mycobacterium:

3. Mycobact. influenzae.
9. Mycobact. diphtheriae.
13. Mycobact. tuberculosis.

M. Streptothrix:

1. Streptothr. bovis.
11. Streptothr. chromogena.
14. Streptothr. farcinica.

N. Leptothrix:

M. Cladothrix:

6. C. intestinalis.

N. Thiothrix:

O. Beggiatoa:

2. HYPHOMYCETES OR MOLDS. In the more severe types of enterocolitis the fecal fields regularly show the presence of this class of micro-organisms. A great deal of work remains to be done among the members of the fungi of this group that are parasitic in the intestinal canal.

Cultural experiments have shown the great importance of these fungi in the pathology of this disease. Their biological processes are usually rapid and intense and the resulting chemical bodies at times extremely toxic both to the mucosa of the intestinal canal and to the general body parenchyma. The enzymes that they use in gaining their livelihood are of great activity, are very diffusible and are so constructed as to act extracellularly. These ferments attack starches, sugars, cellulose, proteins, oils and fats with vigor. The waste products of the growth of these micro-organisms are also important and generally extremely irritating. Their enzymes, under certain conditions, are capable of doing great damage to the tissues of the body that have a fatty structure, especially the nervous system. In other catarrhs their damage arises not so much through toxic action as through mechanical obstruction of the absorbing surfaces of the intestinal mucosa. They very generally may be classed as Fungi Imperfecti.

(a) Trichothecium:

Septate, never forms sporangia, conidiophores not united into definite bodies; conidia and hyphae never pigmented. Conidia double celled, solitary, not in chains; both cells smooth; conidiophores not branched; conidia at tip, never on sides of conidiophores; conidia spherical or pear shaped, two cells often unequal in size; conidiophores long. (Buchanan.)

In sprue in Gram stained fecal preparations the hyphae are positive, deeply stained, unless degeneration has set in, when they are positive and negative in sections or areas. They are septate, the septa being spaced about 5 microns. The stroma stains granularly with here and there punctate nuclear matter. Branching is not noted. The hyphae shows a tendency to bend at the septa in more or less obtuse angles. They occur in felted masses or in twisted strands, strongly resembling silk floss. The length of the hyphae is usually 30–40 microns, the diameter 1.5 to 2.0 microns.

The conidia are double celled, solitary, never in chains.

Both cells are smooth, conidiophores not branched, conidia borne on the tip, never on sides of conidiophores. The cells are pear shaped and are unequal in size; the conidiophores are long, with constrictions spaced about eight times their breadth; the dimensions average 3.6x1.5 microns.

This apparently is a member of the family Mucedinaceae, genus Trichothecium.

(b) Monilia:

Conidia never borne in sporangia; mycelium septate; conidiophores never united into definite bodies; hyphae and conidia never colored or smoky; *conidia one celled*, never multi-celled; conidiophores never sharply differentiated from mycelia, sometimes lacking. Conidia develop by breaking up of hyphae. Conidia develop on definite branches; mycelium well developed and compact.—(Buchanan.)

In the fecal fields, this mold occurs as a rod about 6x1 microns with square ends. The ends are usually Gram positive. The body of the micro-organism is Gram negative with one streak of Gram positive matter along the side. The conidia are cuboidal in form, and are capsulated with unevenly stained protoplasm. Their dimensions are approximately 1x2 microns.

The monilia grow luxuriantly on glucose nutrient agar media. The mycelia are compact, branching, positive or negative to Gram stain, according to their age. They are cuboidal in shape, with dark, Gram positive lines across the ends of the hyphae and many of the hyphae have on the side a Gram positive line, as in the types found in the feces. The hyphae are 4 microns in diameter, septate and branching. Smaller aerial hyphae, the tips breaking into square-end oidia, 4 x 7 microns in diameter, will be noted. The cuboidal structure of the mold is very characteristic. The hyphae and conidia are coated with mucus. Grown under anaerobic conditions, the hyphae and conidia are smaller and approach more nearly to the type found in feces. Considerable gas is produced in growth under these conditions.

(c) Torula:

Family Dematiaceae: conidia never borne in sporangia; septate mycelia; hyphae and conidia are both dark or smoky; conidia never spiral or radiate; mycelium little developed, breaking into oidia, or on short lateral hyphae; conidia in chains easily broken apart into free conidia. If the conidial chains are not easily broken apart, genus Hormiscium. —(Buchanan.)

In the fecal fields torulae when young are strongly Gram positive and take the form of hyphae of medium size, 1.50 to 2.00 x 10 to 30 microns. As they develop, slight indentations appear along the edges. In the shorter ones, consisting of two elements, this gives the micro-organisms a "dumbbell" form. As the chains grow older, the bodies of the conidia become negative with Gram positive dots at either pole. Often a constriction is noted in the middle portion of the conidia, giving a dumbbell form, with negatively staining body and two polar Gram positive bodies. The conidial chain may contain as many as twenty conidia. Occasionally the hyphae of these torulae, in which conidial division has not occurred, may be observed. These are septate, usually Gram negative or delicately positive, with a small positive dot in the central zone of each section.

In glucose media the growth of these torulae is very luxuriant. At the end of four or five days, at room temperature, a fuzzy, slimy growth starts out from the line of feces with which the media is sown. Under the microscope a few hyphae will be observed. These measure 2 x 4 microns to 3 x 50 microns, are usually broader at one end than the other and take a delicate blue tint with Gram stain. Many of the hyphae will be seen dividing into conidia. They are usually found in mycelia, consisting of two or three elements, rarely more. The conidia are fusiform, one celled; capsulated, 1.9 to 2.5 x 3.5 to 7.5 microns, many showing Gram positive polar dots and areas. Neither the conidia nor the hyphae have a mucous coating. With iodine they stain yellow with clear areas.

In certain putrefactions where the presence of fatty acid crystals in the stools is especially marked, this micro-organism is very abundant in the fecal fields and seems to vary in number with the severity of the intestinal condition. The reader is referred to the chapter on Oleic Putrefaction for further information.

(d) Sporotrichum.

Septate, never forming sporangia, conidiophores never united into definite bodies; conidia and hyphae never pigmented; conidia single celled, conidiophores sharply differentiated from the mycelium, conidiophores branched or unbranched, but conidia never forming a terminal head, hyphae never whorled, branched and conidia produced irregularly on lateral conidiophores, never from minute teeth; conidiophores are never upright.—(Buchanan.)

3. SACCHAROMYCETES OR YEASTS:

A host of yeasts gain their livelihood in the contents of the intestinal canal. Whether their presence in the intestines is harmful, or whether they may be regarded as benign, is an open question. We observe, however, that the greater their abundance, the more severe the sickness and also that the predominance of the round-cell type is an unfavorable sign. There has been a great deal of work done with these micro-organisms in connection with the brewing and wine industry, but at present our knowledge of the pathogenic varieties, with the exception of those invading the tissues, is fragmentary.

The position that they may occupy in certain symbiological putrefactions is important. In this connection their presence in many feces from individuals showing a marked increase in acetone production, is interesting.

Many strains are known to produce bodies of extremely disagreeable taste and odor and as materials repulsive to the olfactory system of humanity are not generally well born, either enterally or parenterally, an abundant growth of Saccharomycetes in the intestinal canal is always viewed with suspicion.

CHAPTER III

THE EXAMINATION OF FECES

Laboratory workers very generally regard the examination of fecal matter as disgusting in the extreme. When, however, this subject is approached in a true scientific spirit, these investigations lose their repulsiveness and are no more disagreeable than many other things that must be done in the pursuit of science.

The best container for fecal specimens is a wide-mouthed preserve jar, fitted with the ordinary cover and rubber ring.

For the safety and convenience of the laboratory force, jars with insecure covers, jelly glasses and specimens mixed with water, toilet paper and urine should be refused. It is customary after finishing the examination of a specimen to sterilize with formaldehyde solution. The contents of the jars are then thrown into the watercloset and the jar thoroughly washed. The jars are then broken to avoid the possibility of further use and to save storage space, packed away in barrels to be removed by the ash man at his next visit.

The result of the examination is carefully recorded and the stained microscopical preparations are preserved for future reference in ordinary slide boxes.

Upon the receipt of a specimen the name and date are immediately recorded in the laboratory register and given a number which is pasted upon the container. This number is recorded in the history of the patient, affixed to the specimens taken for preservation and carried in the record of all cultures. The result of the examination is recorded on the history card of the patient under the following heads:

1. Macroscopic.
2. Chemical.

3. Microscopic.
   (a) In water.
   (b) With Lugol's solution.
   (c) With Gram's stain.
   (d) With carbol-thionine stain.
   (e) With iodine stain (full strength Lugol's solution).
   (f) With carbol-fucine stain.

4. The Diagnosis.

Under the heading macroscopic examination, the following facts are recorded:

(a) *The form:* sausage-shaped; scybalous; soft; soft with scybalae; broken; fibrous; spongy; fermenting; creamy; creamy solidifying upon cooling; liquid; liquid with solid portions or scybala; granular; serous; watery; bloody.

(b) *The color:* brown; reddish brown; brownish yellow; yellow-brown; yellow; grayish yellow; gray; light gray or clay color; black; green; olive.

(c) *Mucus:* Absence; presence; amount; jelly-like; glairy; mucopurulent; tapioca-like; masses free or intimately mixed; bloody; bile-stained.

(d) *Gross particles:* presence or absence; soap masses; enteroliths, intestinal sand.

(e) *Parasites:* presence or absence; kind; condition.

(f) *The odor:* aromatic; stinking; butyric; acetic; musty; ammoniacal; ether-like.

Chemical examination:

This includes as a routine measure the tests for blood, for acidity and the estimation of the reaction to Ehrlich's aldehyde.

For occult blood the benzidin, acetic acid, hydrogen peroxide test is usually employed as the most convenient. The aloin or guiac test may be used, if preferred.

All of these tests are valueless unless that patient has been

on an iron free diet for several days. Therefore all kinds of meat, green vegetables and medicines containing iron should be excluded from the dietary for at least forty-eight hours before taking the specimen.

This test may be performed in a test tube. It is more convenient, however, to use a shallow glass vessel, such as a Petri dish, as in the case of very delicate reactions it is convenient to examine with the microscope for particles positive to benzidin.

The benzidin should be fresh and the acetic acid pure. Contamination with any vessels stained with any preparation of iron should be carefully avoided. A small piece of the feces under examination is placed in a Petri dish, a little to one side of the centre. This is mixed with an equal amount of hydrogen peroxide. A small amount of benzidin is placed on the opposite side of the dish and mixed with two or three drops of concentrated acetic acid. Then with a clean glass rod the two mixtures are brought in contact. If blood is present in any amount the customary green tint will be observed at the juncture of the feces, hydrogen peroxide mixture and the benzidin acetic acid solution. If no color is apparent to the naked eye, the specimen may be examined with the microscope for the presence of green-colored particles, denoting the presence of blood in minute amounts.

The reaction to litmus is then recorded. For the estimation of total acidity or for total alkalinity, one gramme of the feces is emulsified with 9 c.c. of distilled water. The reaction of this is estimated by either $\frac{n}{10}$ HCl or $\frac{n}{10}$ NaOH, as the case may require, using phenolphthalein as an indicator.

The estimation of the value of the response to Ehrlich's aldehyde is of great importance. A comparison of the strength of this reaction in the feces with that of the urine gives an absolute value for the efficiency of the liver. The following is the process: A clean beaker is first weighed and the weight recorded. A mass of the fecal matter under examination is spread out in a thin layer over the bottom of the beaker, evaporated to dryness on the hot-plate and

the beaker and contents weighed while still warm. The weight of the beaker and dried contents, less the weight of the beaker alone, is then multiplied by 100 and cubic centimetres of water added to equal this amount. The mixture is then heated and stirred until solution takes place, drawn into the 1 c.c. roller pipette and the aldehyde solution No. 2 titrated, as is described in the chapter on the Urine.

For example:

Weight of beaker and dried feces less
weight of beaker 0.45 grammes,
Times 100, equals 45 c.c. of H_2O added.

Under titration 0.16 c.c. of this solution causes 5 c.c. aldehyde solution No. 2 to show a pink fluorescence. Multiply by two for value of 10 c.c. of aldehyde solution. Result 0.32 c.c. is the aldehyde value of the feces.

The proteolytic and amylolytic activity of the feces may be estimated with Mett's tubes, egg albumen and starch paste. Exhaustive investigations upon the enzymes present in the feces conducted in my laboratory have shown very little of practical value.

Extensive chemical process for the quantitative estimation of mucus, mucine, fats, proteins, carbohydrates; oleic, butyric, acetic, diacetic, formic, propionic, oxalic and other acids and their salts; indol, phenol, skatol and various complex products of bacterial growth and biological activity may be followed out, if time permits.

The specimen is then prepared for examination with the microscope.

A thin emulsion of feces and plain water or decinormal salt solution is made, mounted on several slides, and carefully searched for the eggs, oncospherae or embryos of the intestinal parasites. This is a very important procedure and its omission may lead to very embarrassing situations.

One to six slides are prepared with weak Lugol's solution as a staining fluid and examined wet.

Formula: Dilute Lugol's Solution.
Water 50 c.c.
KI 0.5 grammes.
Lugol's Sol. 5 c.c.

A very speedy and convenient method is the following, as it avoids the use of beakers or other containers. A drop or two of dilute Lugol's solution is placed on a clean glass slide and mixed with a small particle of the feces under examination with the aid of a small wood applicator. This can be burned when the operation is completed. Care must be taken not to have too strong a mixture of feces and solution, yet is it convenient to have one portion of the specimen rather thick to aid in the search for fat needles and fat sheaves. The mixture had better be spread out in a long, narrow strip over the slide, so that the cover glass will not become wet on the upper surface and soil the objective of the microscope.

The preparation is first examined for free starch and, if present, the appearance and kind, whether potato, wheat, oat, etc., recorded. Starch granules are stained blue. A search is next made for muscle fasciculi. These will be with or without striae and in either case the fact should be noted. Crystals should next be searched for. These may be fatty, triple phosphate, oxalate of lime, Charcot-Leyden, iron, bismuth or the residue of some drug administered to the patient. The yeasts are next examined and their character, whether oval or round, and the relative abundance, recorded. The vegetable detritus is examined and its description recorded. Finally a search for amoebae is made.

Four cover glass preparations are then made, two thin and two thick. One of the thin preparations is stained after the method of Gram, the other is stained with carbol-thionine. One thick one is stained with full strength Lugol's solution, the other with carbol-fuchsine.

Gram's Method.

Three drops of aniline oil are shaken with 10 c.c. of water and filtered. Five drops of a saturated alcoholic solution

of gentian violet is added to the aniline water. The specimen is stained with this solution for three minutes. The stain is poured off and without washing is flooded with Lugol's solution, which is allowed to act for two minutes, decolorized with absolute alcohol, counter-stained with Bismarck brown, dried and mounted in Canada balsam.

The Carbolic Thionine Blue (Nicolle) is prepared as follows:

Formula: Thionine blue 1.0 gramme
Carbolic acid 2.5 grammes
Distilled water 100 c.c.
Filter.

Before using, dilute with equal quantity of distilled water and again filter.

Stain the specimen fifteen minutes with the above, wash thoroughly and soak in tap water for one half hour.

In this preparation the alkali forming organisms will be stained dark blue, the acid producing violet.

The thick film is then stained for fifteen minutes with Lugol's solution, carefully washed to avoid the presence of potassium iodide crystals in the finished preparation and mounted in Canada balsam. The other is stained with hot carbol-fuchsine, decolorized with ten per cent nitric acid alcohol and counter-stained with methyl blue. Carbol-fuchsine solution does not keep well and we find it a great convenience in our laboratory to keep phenol solution and the fuchsine solution separate. The method of staining is as follows:

Upon one corner of the smear, a small drop of concentrated phenol solution (liquefied crystals) is placed. One drop of alcohol is then added to complete the solution of the phenol. Six drops of water and one drop of a saturated alcoholic solution of fuchsine are placed on the smear. This is heated until the liquid begins to steam, allowed to stand for ten minutes, decolorized with nitric acid alcohol, counter-stained with methyl blue, dried and mounted in Canada balsam.

Micro-organisms are never named in the history card unless a full identification through cultural methods has been

carried out. The record of the description and not of the name has been found of the greatest convenience. As our knowledge of the micro-organisms that are parasitical in the intestinal contents grows, records and specimens can be gone over again and very useful information acquired from the study.

The description of the various types, however, is recorded very fully and covers the following points:

(a) The groups: streptococcus, micrococcus, sarcina, planococcus, planosarcina, bacterium, bacillus, pseudomona, microspira, spirillum, mycobacterium, streptothrix, leptothrix, cladothrix, thiothrix, beggiatoa, molds, yeasts.

(b) Characteristics of form: long, short, straight, curved, dimensions in microns, square or round ends, oval, diplococci-like, dumbbell-like, clavate, cuneate, capsulated, non-capsulated, growing in chains, growing in masses, filamentous, hyphae-like, septate or non-septate.

(c) Sporulation: equatorial, polar, bipolar, chlamydospore type, conidia-like, stained or unstained, round or oval, discrete or in masses, the size of the spores in microns.

(d) Staining: Gram positive or negative, lightly or intensely, evenly, granularly, punctately, irregularly, sectionally.

(e) The growth: luxuriant or scanty.

To avoid illness among the laboratory assistants, the usual safeguards against infection should be thoroughly enforced, as many feces contain organisms that are pathogenic in a greater or less degree. Bichloride or carbolic solutions should be constantly at hand and great care in the use of laboratory towels and utensils should be taken. In the case of the incubator, a ventilating tube leading to the chimney is a great convenience, especially if the laboratory is in immediate connection with other rooms, and should be supplied when possible.

CHAPTER IV

THE EXAMINATION OF URINE

The study of the urine in enterocolitis gives much valuable information. When properly and carefully performed a diagnosis can be made in many instances by this means alone, but much better results will be obtained if the urinalyses and the fecal examinations are compared and the findings in each carefully verified.

The customary routine examination of the urine is first completed and recorded in the patient's history. This investigation includes the color, the odor, the cloudiness, the specific gravity, the amount of albumen or its absence, a quantitative estimation of the reduction of Fehling's solution, the amount of chlorides and phosphates, the reaction to litmus and microscopically, a thorough search for casts, epithelium, pus and crystals.

The chemical bodies that will be hereafter referred to as the indices of intestinal putrefaction are next taken up in order. At the present writing they are classed as follows:

Indican blue,
Indican red,
Acetone,
Diacetic acid,
Oxalic acid,
Uric acid,
Ammonia,
The body responding to dimethylaminobenzaldehyde.

The following test for indoxyl has proven the most convenient and reliable. The reagents required are chemically pure strong hydrochloric acid, a 0.5 per cent solution of potassium permanganate and chloroform. The appa-

ratus required is very simple—a 25 c.c. glass-stoppered graduated cylinder and a 1 c.c. pipette. The cylinder is first filled to the 5 c.c. mark with urine, then to the 10 c.c. mark with hydrochloric acid and 1 c.c. of chloroform added by means of the pipette. The potassium permanganate should be added a drop at a time and the tube well shaken between each addition, until upon standing the chloroform ceases to grow darker in shade. The two most frequent errors to be guarded against are the too rapid addition of the reducing agent, i.e., the permanganate solution, and insufficient shaking. The depth of the blue or red color of the chloroform is compared with that of the color-scale and the result recorded. In urines showing the mixed types the proportions of blue and red are estimated in comparison with the scales.

For the sake of the accuracy of this test, as well as in the case of any other of the tests for the putrefactive indices, all administration of medicinal agents should be suspended for at least twenty-four hours before the urine is taken.

The presence of acetone is determined by Lieben's or by Gunning's test. If the patient has not taken alcohol the former is preferred. The urine should be as fresh as possible and the amount taken for testing is 50 c.c. An Erlenmeyer flask, fitted with a rubber cork and a bent glass tube of proper length to reach within a centimetre or so of the bottom of a test tube surrounded by cold water, is used for distillation. The process must not be carried on too rapidly. The distillate is then treated with a drop or so of Lugol's solution and decinormal sodium hydrate solution added until the color of the iodine has been discharged. The strength of the reaction is expressed in plus marks, thus: + 0000 represents a trace or a slight odor of iodoform upon heating the mixture, ++ 000 an abundance of crystals when examined microscopically yet not visible to the naked eye, +++ 00 a visible precipitate, ++++0 a considerable precipitate, and finally +++++ a reaction so strong as to produce an immediate cloudiness upon adding the sodium hydrate solution.

If greater accuracy is desired, by slow distillation, using ice water in the condenser, collecting the iodoform on a filter, washing first with very dilute hydrochloric acid and then with distilled water, and finally weighing with the analytical balance, the results may be expressed in milligrammes of iodoform per 100 c.c. of urine, and this figure used in judging the progress of the illness.

If Gunning's test is used the tube should never be heated until the black precipitate of nitrogen iodide has entirely disappeared.

The Gerhardt's or Bordeau red reaction is used in testing for diacetic acid. It is absolutely necessary that the urine be perfectly fresh. In moderately cool weather, if the morning urine can be examined before noon of the same day, the test may be depended upon for accuracy, but during the summer months it is much safer to have the patient pass urine in the office and the examination made immediately. All medicines should be omitted for twenty-four hours before the urine to be tested is taken. The strength of the reaction is recorded in plus marks, with a statement of the character of the reaction. If the test is negative the symbol 00000 is used; if a light brown shade is obtained Bn+0000; darker shades of brown are expressed by extra plus marks as in the case of the test for acetone. If the test gives absolutely no red shade, the symbol By00000 is added, while extra plus symbols are added to express reactions of increasing intensity until the deepest Burgundy reactions are recorded as By+++++. An admixture of the two colors may also be recorded by these fomulae if desired; for example, Bn++000By+0000 would mean a moderately light brown reaction showing a faint red tint. If the precipitate of ferric phosphate obscures the test it may be removed by filtration.

The brown shades, outside of the question of the presence of diacetic acid, are also valuable in the estimation of the amount of organic acids and acid salts excreted by the kidneys.

Oxalic acid always occurs as calcium oxalate when present in the urine. After centrifuging and examining under the

microscope, the number of crystals of this compound may be expressed as few, numerous, abundant or extremely abundant, as the case may be.

Their abundance, however, may be much better expressed in the number per cubic millimetre of urine times the average diameter in microns of the bases of the crystals.

The presence of uric acid crystals is always noted in the record of the urinalysis, but as a general thing the quantitative estimation of the amount of this acid is not carried out.

The presence of ammonium-magnesium phosphate crystals in any abundance usually means that a putrefactive process with the production of ammonia is present in the intestinal canal. The source of this ammonia should always be confirmed by comparison with the fecal analysis. If the latter shows the reaction for free ammonia or the presence of triple phosphate crystals, the ammoniacal type of putrefaction may be diagnosed.

The test with dimethylaminobenzaldehyde is of great value. There are two methods of recording this test—by the use of the color scale, and by titration. In both methods the following formula is used:

Ehrlich's Aldehyde Solution, No. 1.	
Para-dimethylaminobenzaldehyde	15 grammes.
10% Solution sulphuric acid	300 c.c.

This gives a clear, amber-colored solution, which upon standing takes on a greenish tint and upon boiling has an aromatic odor reminding one of new-mown hay. This solution improves with age.

To perform the test, five cubic centimetres of the urine are brought to the boiling point and the solution added drop by drop until no further deepening of the red tint can be obtained. While the mixture is still hot, the shade is compared with the color scale and the result recorded. A modification of the ordinary hemoglobinometer may also be used. In either case the strength of the reaction will be expressed in a percentage based upon the color scale used in estimation of the hemoglobin in the blood. The depth of the color may

also be recorded in plus marks, 00000 representing an absence of any reaction, +++++ the deepest red tints and the intermediate shades by combinations of the two symbols.

The use of color scales in the laboratory is hedged about with inaccuracy, and it has been found that the titration method, especially in instances where this test has been used in checking the dietary of the patient, gives much more uniform results.

The apparatus required for the proper performance of this operation consists of a one cubic centimetre pipette graduated in 0.01 c.c., a roller pinch-cock with rubber tubing, a 10 c.c. graduated cylinder, test tubes and Bunsen burner. There is nothing especially novel about this apparatus excepting the roller pinch-cock, and upon the smoothness of the operation of the latter the success or failure of the test will in a great measure depend. This consists of two metal rollers and the containing frame. The rollers are 1.5 centimetres in diameter and 2 centimetres on the face. The surface of the first roller is scored parallel to its axis with a hacksaw, in order that the rubber tubing that is to pass between them may not slip. The scorings have a depth of 0.5 millimetres and are spaced 1 millimetre from edge to edge. A knurled wheel is fitted to the shaft of this roller and the outer edge of this wheel is supplied with a small crank handle. The second roller is left smooth and revolves on a shaft constructed to engage the yoke mentioned below. The frame is constructed of 2 millimetre flat stock, enough length being allowed for securing the apparatus firmly to its support, bored and slotted so that the scored roller and knurled head has a good running fit, and the smooth roller may be brought towards or away from its companion as may be required in the adjustment of the apparatus. A yoke and adjustment screw is then fitted to impart motion to the shaft of the smooth roller in such a manner that the surfaces of both rollers will always remain parallel. With rubber tubing of proper length any pipette may be operated with this device and it has been found very convenient in measuring infected solutions.

The following formula is used in making this determination:

Ehrlich's Aldehyde Solution No. 2.	
Aldehyde solution No. 1	10 c.c.
Distilled water	90 c.c.

Five cubic centimetres of this solution are raised to boiling temperature in a test tube. When the surface of this solution is observed at an angle of 45°, the base of the tube resting on a white surface, a delicate green color will be noted. If this color is not present, or if a red tint is noted before the addition of the urine, some of the utensils are not clean and the operation should be repeated with fresh solution. The pipette is then filled to the zero line with the urine under examination and any excess of urine removed from its tip with blotting paper. The solution is kept at the boiling point during the titration and urine added drop by drop until the surface of the mixture when observed at an angle af 45° assumes a delicate pink shade. The amount of urine used multiplied by two is the value usually used in expressing the strength of the reaction. This figure, of course, is inversely proportionate.

The matter of the dimethylaminobenzaldehyde reaction in both urine and feces has been under discussion for the past fourteen years without arriving at any definite conclusions. Skatol, urobilogen and glycosamin have by various authorities been mentioned as the bodies responsible for this reaction. Professor Herter first noted that the urines from individuals suffering from the most excessive intestinal putrefactions were especially liable to give the most pronounced reactions. Among my own patients I have learned to associate this reaction with severe constitutional symptoms and to regard a lessening of its strength a most favorable sign, an increase, as foreboding disaster.

The color of the urine has little influence upon the strength of this reaction. As many dark urines give a very delicate reaction, while in others of a pale straw tint it is very well marked, apparently the urinary pigments cannot be held responsible.

Urines that gave a reaction of 0.10 and that were kept in the light and shaken frequently did not give any increase in its strength. Others were extracted with ether to remove urobilogen and both the ether and the residual urine tested. The aldehyde value of the urine remained unchanged. The ethereal extract, evaporated to dryness and dissolved in alcohol, was negative.

The question of the protein split products was taken up as a possible source of this reaction. Egg white was precipitated by 95% alcohol, filtered, the filter washed thoroughly with ether, dried and powdered. This was digested in two per cent sodium hydrate absolute alcohol, after the method of Vaughn, and both the alcohol and the solid remaining portion tested with aldehyde. The alcoholic extract was positive. The solid portion took on a pink shade when examined by light reflected from the surface of the particles, while by transmitted light they were a delicate gray. Specimens of the untreated egg white were negative to aldehyde.

In the case of dessicated thyroid gland the soluble portion was negative, the insoluble portion gave a dark red reaction.

The sugars were uniformly negative.

Cultures of mixed fecal bacteria were positive. Broths when freshly prepared were negative. When putrefaction had begun they showed a marked reaction to aldehyde.

Witt's peptone was positive, as well as other beef preparations, advertised as "predigested." Blood was negative, the scales from psoriasis were negative. Specimens of Witt's peptone were extracted with absolute alcohol, filtered and dried. The filter was positive, the filtrate was negative to aldehyde. Typhoid bacterins were negative.

Fluid extract of ergot was positive. Vaughn has called attention to the presence of certain protein split products in claviceps purpurea, so this reaction was not surprising.

Ergotoxin, a trade name given to an ergot derivative, gave a positive time reaction. Upon first adding the aldehyde solution no change in color was noted upon boiling. After standing a few minutes the mixture changed to a pronounced purple color, which deepened with further boiling.

A specimen of ernutin, another ergot derivative, gave the same reaction as ergotoxin.

When ernutin was treated with boiling ten per cent H_2SO_4 and allowed to stand twenty-four hours the reaction with aldehyde in the hot was very sharp and distinct.

Specimens of ergamin, said to be Beta-iminazolylethylamin, were negative. When digested with ten per cent H_2SO^4 for several days this drug was still negative.

Specimens of pancreatic gland substance, combined with bile salts were slightly positive.

Fel bovis was negative.

Various preparations containing bile salts were negative.

It was noted that during the titration of the aldehyde solution that at the instant the change in color took place, there was also an ebullition of some gas, probably hydrogen.

Noting the fact that this reaction depended upon a change in color of the reagent from green to red, and that this apparently took place through an increase in the size of the molecule of the aldehyde, the question of oxidation came to mind. The aldehyde solution was consequently tested for response to oxidizing compounds. Hydrogen peroxide even in very weak solutions gave a marked positive reaction. Magnesium peroxide also gave a very strong positive reaction. Potassium chlorate, potassium permanganate, nitric acid, bismuth subnitrate and zinc oxide were negative. Fuming nitric acid (nitrous acid) in the cold produced a yellow reaction with the escape of gas; upon heating, the mixture grew darker and finally assumed a reddish brown color.

Two forms of fecal fields are regularly found in patients whose urine and feces give a marked reaction to aldehyde. The first shows a very luxuriant growth of short, plump, round end, Gram negative rods, which are probably the bacillus of Mellanby and Twort. In the second these bacilli are absent and in their place hyphae and conidia are found in abundnace. The bacilli probably produce in their growth a near chemical neighbor of beta-iminazolylethylamin that is responsible for this reaction; and cultures from such feces are also strongly positive to aldehyde. In the case of the

molds the evidence pointing to the production of *β-i* is not so clear. In view of the fact that the response of aldehyde to oxidizing agents, even in minute amounts, is especially prompt, the presence of an oxidase, the result of the biological activity of intestinal molds, may possibly account for this reaction in case the bacilli are absent from the fecal fields.

If the liver is efficient this body will not appear in the urine in an amount proportionate to its abundance in the feces. It will be reduced in its passage through that organ to such an extent as to no longer respond to aldehyde.

Judging from urinalyses in subjects undergoing typhoid immunization, this reaction does not appear in the urine during the course of parenteral protein digestion, at least in normal individuals.

In the urine of sufferers from epilepsy the response to aldehyde is remarkably strong and more especially so when an attack is imminent. The presence of a value as low as 0.05 is generally followed by a convulsion within twenty-four hours.

The test for urobilin is useful in estimating the amount of the destruction of erythrocytes that is taking place. Schlesinger's method will be found the most convenient. The following reagents are required:

1. Zinc Acetate Alcohol.
   Absolute alcohol 100 c.c.
   Zinc acetate to saturation.
2. Lugol's Solution.

To the unfiltered urine an equal amount of the zinc acetate alcohol is added. This is shaken and a few drops of Lugol's solution added. The mixture is then filtered and the degree of fluorescence of the filtrate recorded in plus marks.

CHAPTER V

THE INDOLIC TYPE OF INTESTINAL PUTREFACTION

In this type, the onset of the primary stage may be very acute with a diagnosis varying from enteric grippe to paratyphoid fever.

In children, we often meet with attacks of fever lasting from a few days to several weeks, in which the urine gives a very strong reaction for indican. The sera of these children do not agglutinate the typhoid bacillus. This is usually the beginning of an enterocolitis with this type of intestinal putrefaction and the gravity of this condition is not given the attention it deserves. This type often develops in the course of a chronic enterocolitis, which in the beginning showed another type of putrefaction.

Infection in these cases may take place in two ways: by way of the mouth through the agency of those vegetables which are grown in intimate contact with the soil, such as radishes, lettuce and celery, by contact, either direct or indirect, with sources of infection in street dust, water closets, door handles and car straps; or by infections extending upward from the colon. In the latter form of infection, which may be called the ascending form, insufficiency of the intestinal valves, certain ptoses of the intestines or a weakness of peristalsis, facilitating the progress of bacteria from one portion of the intestine to another, may be assumed.

Certain families seem to have a special lack of resistance of the intestines against this form of bacterial invasion.

The symptoms of this condition may be considered, according to the various stages, viz. the primary stage, if such exists; the secondary stage and the tertiary stage.

The symptoms in the primary stage may range in severity

from those causing little trouble, such as suddenly developing headaches and mild abdominal symptoms, to severe febrile attacks, confining the patient to bed with high temperature. These symptoms may last from three days to two weeks, often giving a clinical picture which strongly resembles a mild typhoid infection. The true nature of these symptoms is apt to be overlooked, but an examination of the urine will show the presence of indican in greater or less amounts.

In the secondary stage, the symptoms are not generally constant. Attacks of dyspepsia, so-called indigestion, epigastric pressure or distress, lasting for a fortnight perhaps with a remission of about a month and then a return of symptoms, is a suspicious syndrome. In this stage the symptoms are confined to the abdominal organs and are described as distress or discomfort, burning, pyrosis, with or without constipation or diarrhoea. These are laid at the door of indigestion or dyspepsia, nervous being a most favorite prefix. As a collateral diagnosis, the patient may add chronic appendicitis, chronic cholecystitis and intestinal indigestion to the list of his complaints. Obscure disturbances of nutrition and metabolism are at times prominent.

The tongue is very generally coated, especially in the morning, and the breath has a fecal odor. For the relief of this the patients resort to habitual catharsis very early in this stage. While cathartics give a marked temporary relief in this type, no permanent improvement is ever noted from their use.

The circulatory system shows functional disturbances very early in this stage and in many instances the patient believes that he is the victim of some severe and fatal cardiac disorder. Palpitation, tachycardia, fainting spells and dyspnoea are frequent complaints.

The nervous system is also the seat of many functional disturbances and such disorders as headache, either frequent or habitual, insomnia, vertigo, neurasthenia are very apt to be present. Migraine, however, is rare in this type of putrefaction and its presence always leads one to suspect some of the more severe acid types as its cause.

The specialized organs, the eye, the ear and the genital system may also show some disturbance of function. Many of these patients complain of impotence or sterility and it is interesting to note that, in common with the other forms of chronic infection, the birth rate among these sufferers is low and the offspring of poor vitality.

These patients usually pass the secondary stage in an anemic condition. Their strength is poor and even if there is no complaint of its loss, still their endurance is very unsatisfactory, both for physical and mental work.

In this stage the physical examination shows little of moment. The liver is neither larger nor smaller than normal; the stomach is normal to palpation. The colon may be distended but not tender. The position of the abdominal viscera will correspond to the patient's habitus. Physical signs of disease in other organs will be lacking. These individuals, on the other hand, give the examining physician the impression that they are ill.

The stomach contents show little of interest. There is usually, however, a hyperchlorhydria but no evidence of organic gastric disease.

The urine, in the secondary stage, will show no evidence of kidney involvement. The strength of the indoxyl reaction, however, will give very valuable information concerning the severity of the intestinal condition that is slowly but surely undermining the patient's health.

The gross appearance of the feces is usually fairly normal. They may be formed, soft or liquid, their condition depending upon the kind and amount of cathartics the patient is using. There is little mucus; no gross particles; the color is dark; the odor not abnormal. The reaction is alkaline. With Lugol's solution free starch is absent, muscle fasciculi and crystals are absent, yeast cells are few in number and oval in type.

It is in the preparations stained according to the method of Gram that we find the most valuable information concerning this type of putrefaction. Upon the first glance through the microscope, the great prominence of the blue

or Gram positive organisms will be noted. The largest of these will be a bacillus, capsulated, growing end to end in pairs. This will be recognized as the bacterium Welchii. These are not usually very abundant and their growth, in the milder grades of this type of putrefaction, is not luxuriant. The next most abundant form will be the bacillus bifidus—medium sized, irregularly contoured rods, often of headlet form, at times bifurcated. The bacillus putrificus is absent unless the disease has been in progress for some time. Positive micrococci are very rarely observed, as in the simple indolic type ulcerative processes are rare. An occasional clostridium may be observed.

In the negative field the small number of bacilli of the colon type will be noted. In the lighter grades their number may be considerable, but in the more severe putrefactions they will be absent. The severity of the catarrhal process may be estimated by their number—the less abundant they are the more severe the disease. The fine, negative bacilli of the liquefaciens ilei type may be present when the stools are soft in consistency. Short, fat rods without capsules are usually absent. It is rare to find hyphae and spores except in the more severe grades of putrefaction and then only when the disease has been in existence for some years.

The preparations stained with thionine show the same micro-organisms mentioned above.

The iodine field is generally negative. In some specimens, however, a few clostridia may be noted.

The blood shows little of moment in the average patient. In the latter part of the secondary stage, the Hb. value begins to diminish and in some instances the number of red cells are reduced. An anemia of the pernicious type places the individual in the tertiary stage of the disease. The fact must be borne in mind, that the blood may be the point of attack of this toxin without other organs showing organic lesions.

The secondary stage may continue for fifteen to thirty years without greatly impairing the patient's capacity for work. The sufferers are much more likely to regard themselves as more uncomfortable than sick.

In the tertiary stage abdominal symptoms, per se, form a very unimportant part of the clinical picture. Constipation may be present or often an intermittent diarrhoea The symptoms referred to the intestinal canal are not so impressive as those that arise from organic disease of the heart, kidneys, liver or other organs that have begun to find their burden too great.

Various forms of muscle and joint lesions are common in the tertiary stage, such as myositis, osteoarthritis and rheumatoid arthritis and the finger joints usually are the seat of calcareous deposits.

In the nervous system organic lesions are met with of various grades of severity, leading to psychoses, neuralgia, low grades of paralysis and disturbances of function.

In certain sufferers from this tpye of putrefaction a condition develops that may best be described by the term presenility. Outside of a low grade anemia, great depression and loss of strength and endurance they show very few symptoms. The physical examination is usually negative, the blood pressure normal or below normal, but besides a marked indicanuria little can be found. They become a burden to themselves and their family and usually die from some intercurrent disease.

CHAPTER VI

THE SACCHAROBUTYRIC TYPE OF INTESTINAL PUTREFACTION

"This form of intestinal derangement is characterized by a chronic putrefactive process (having its seat mainly in the large intestine and lower ilium) and due to the action of very large numbers of strictly anerobic butyric-acid producing bacteria, capable of multiplying by means of spore formation."—(Herter.)

The onset of the primary stage of this type of putrefaction is more generally gradual than sudden. There are patients, however, who date their trouble from some acute illness, although the presence of a true saccharobutyric type at that time cannot, of course, be determined.

The secondary stage is of long duration. There is not usually a very marked departure from a condition of fair health, but the patient's most frequent complaint is one of indigestion, dyspepsia or of other symptoms due to the irritation resulting from the presence of ammonium butyrate in the intestinal canal. Constipation is usually the rule, but often a diarrhoea may be present, which in some instances may prove refractory to treatment. Considerable burning accompanies this acute disturbance. The constipation will follow the usual course, beginning as the atonic type, passing into the spastic stage, but rarely, in uncomplicated cases, reaching the point of stercoraceous diarrhoea. Often the amount of flatus is greatly increased, the composition of which will be found upon analysis to be hydrogen and nitrogen. The flatus is usually of very little odor, the aromatic character of normal human feces and flatus will be absent. Occasionally a mild odor of old cheese may be noted.

The mouth is frequently sore and the tongue, from the tendency of the superficial epithelial layer to exfoliate rapidly, is usually bright red. The tongue may be glazed and its surface is often cracked. In very severe conditions the skin may also be extremely irritable, especially sensitive to light and wind, and in many of these people the exposed surfaces of the body are in a constant state of inflammation during the summer months. In some people the subcutaneous and the submucous tissue is liable to attacks of oedema of very severe grade, the so-called Quincke's oedema, and the possibility of this swelling involving the glottis must always be borne in mind. The muscular system is often the seat of rheumatic pains of greater or less severity.

In the nervous system, outside of some vertigo or headache, nothing very alarming is present.

In the circulatory systems very few disturbances are found excepting slight irregularities in the heart's action and palpitation.

If the liver is still equal to its task, very little trouble will be noted outside of the intestines. When the patient's diet has been of such nature as to encourage this type of putrefaction, acute disturbances will result with an increased production of butyric acid. The liver is then found swollen and often tender to pressure.

A mild grade of anemia is very common.

The patients often complain of a moderate loss of strength, often progressive, and their endurance is generally so poor that the completion of the day's work becomes extremely burdensome.

The onset of the tertiary stage is not violent, gradually the signs and symptoms of degeneration in few or many organs make their appearance and the patient slowly sinks under the burden that the wreck of some specialized organ imposes.

Examination of the gastric contents usually shows very little of a pathogenic nature.

It is in the examination of feces, however, that the most valuable information is found.

Macroscopically the stools may be of large or small calibre, the color varying from deep brown to reddish brown, except at times of diarrhoea, when they approach a light yellow tint. They often contain mucus.

Stained with dilute Lugol's solution, free starch is occasionally found, but muscle fasciculi with striae are uniformly absent. Clostridia may be abundant. With Gram's stain the abundance of positive diplobacilli and biscuit-shaped bacilli with strongly refracting capsules will be noted. The smaller bacilli, often headlet in type, will be very numerous and the large and small filaments will be absent. Large, lemon-shaped clostridia will be seen, growing in groups and sporulating. In the negative field the presence or absence of the colon bacillus and, if present, their numbers will give very valuable information as to the severity of the infection and the length of its standing. The greater the number of bacteria of the colon type found in the fecal fields, the lighter and more amenable to treatment will be this condition. The presence of streptococci, staphylococci is always a bad sign and denotes the presence of ulcerative processes in the small intestine or in the colon. With thionine stain most of the micro-organisms will take a violet shade, especially the B. Welchii. Very few will take a blue shade in this type of putrefaction. The iodine fields will generally show an abundance of the clostridia of Prazmowsky, sporulating and of very luxuriant growth. The field will be mildly Gram positive to absolutely positive according to the severity of the condition.

The urine will be acid and usually of high specific gravity. Blue indican is never present in simple cases, although indican red will be found in small amounts. Diacetic acid and acetone are uniformly absent. The test with Ehrlich's aldehyde is usually negative. In the tertiary stage albumen and casts often make their appearnace; in the primary and secondary stages, the urine is microscopically negative.

This is the most common type of intestinal putrefaction as well as the least fatal. The duration of many cases must be measured in decades, not in years. In many individuals

with the milder grades little inconvenience is suffered. They may realize that they have a liver, that certain articles of food are better left uneaten. Their general health, however, is not markedly impaired and they often attain a ripe old age and die from some intercurrent disease without ever entering the tertiary stage of enterocolitis with the saccharobutyric type of intestinal putrefaction.

The liver is the organ most liable to attack. Through years of overwork in oxidizing the acid products of the disease a cirrhosis finally develops. This in turn, through the pressure of the contracting fibrous tissue, further damages the stroma until finally the patient ends his days on a milk diet. This type is especially common in individuals who have overindulged in severe physical exercise, such as the professional football player, the heavy weight lifter, the iron bar bender, and others who earn their living by feats of strength and have thereby injured the oxidizing power of their liver. In such individuals the probability of a complete recovery is very remote.

CHAPTER VII

THE ACETIC TYPE OF INTESTINAL PUTREFACTION

This type is characterized by the production of β-oxybutyric acid, diacetic acid and acetone, either singly or in combination, in the intestinal canal.

The primary stage is not usually marked by symptoms severe enough to confine the patient to bed, and is passed with the diagnosis of overwork, in need of a tonic, or some of the terms so often used in slipshod diagnosis is applied to it. At this period the tongue is usually coated, some abdominal distress may be present and occasionally constipation or diarrhoea of mild grade. In other words, the patient suddenly finds his vitality reduced from some obscure cause. Recovery in this stage may be spontaneous and complete.

If this condition advances to the secondary stage, the patient begins to notice an increasing loss of strength and vigor. With this he begins to develop a pallid complexion, notices that his tongue is habitually coated, finds great relief from the more or less constant use of cathartics, but rarely does he report any great diminution of his appetite. As he advances in this stage he complains more and more of intense thirst. The abdominal symptoms in uncomplicated cases are usually of little moment. This stage may run on for several years or even decades.

The tertiary stage may come like a thief in the night and the individual suddenly awakens to the fact that his days are numbered. On the other hand, the most intense abdominal symptoms of very alarming character may be present. Occasionally the nervous system bears the brunt of the attack, and convulsive seizures or a comatose condition will develop without warning. There is always the possibility that, in

the latter instance, the individual may die without regaining consciousness.

Acetone production is a common occurrence in persons who consider themselves in fair bodily health. In this case, however, we may assume the presence of a mild intestinal catarrh that has so reduced the strength of intestinal antiseptic as to render putrefaction possible, without being of sufficient severity to cause any local symptoms. This acetone production may be either continuous or intermittent, varying in severity as the person's intestinal resistance may vary from day to day, or with the character and condition of the food ingested.

The anemias accompanying this type of putrefaction with the production of acetone alone are often severe and the destruction of erythrocytes very marked.

In many instances acetone may be present in the circulation in such amounts as to impart a pronounced odor to the breath without any marked departure from fair health.

In diacetic acid, however, we have to deal with a much more potent poison. Rarely is this body found in the urine without toxic symptoms referred to various organs of the body occupying a very prominent position in the syndrome. The liver is very generally the first to feel the attack of this poison and is found swollen, the edge round and firm and usually sensitive to pressure. The liver may be regarded as a filter placed between the circulation through the intestinal wall and the general blood stream. One of its most important functions is to promptly remove any acids or toxic bodies from the blood that passes through it and to hydrolyse them into simpler and less irritating chemical bodies. These poisonous bodies, unless they are of such stable composition as to be incapable of oxidation in the animal cells, are, in the case of most of the toxins absorbed from the intestinal contents, reduced to carbon dioxide and water. This chemical process is similar to that which takes place when sarcolactic acid is washed out of the muscular system and brought to the liver for disposal. Therefore, when a severe acid putrefaction is present in the intestinal canal, an increased excre-

tion of carbon dioxid is present, provided a requisite amount of oxygen is supplied to the liver cells. While the liver is equal to the task, the constitutional symptoms are few in number and of little moment. When, however, that great biochemical apparatus begins to find the task too heavy, when the members of the fatty acid series begin to appear in the general circulation and the burden of oxidation is thrown upon the parenteral and parhepatic cells, then the patient first realizes that there is a marked departure from normal conditions. Strange to say, he rarely complains of symptoms referred to the seat of trouble; on the contrary, it is the heart, the nervous system, the kidneys, or a general feeling of weakness that first attracts his attention.

β-oxybutyric acid is never found in the urine unless the function of the liver is impaired or exhausted. The complete reduction of one molecule of β-oxybutyric acid requires nine atoms of oxygen. Thus:

$$CH_3–CHOH–CH_2–COOH + 9\ O = 4\ CO_2 + 4\ H_2O,$$

in the case of acetone eight are required:

$$CH_3–CO–CH_3 + 8\ O = 3\ CO_2 + 3\ H_2O,$$

while with diacetic acid eight also are required:

$$CH_3–CO–CH_2–COOH + 8\ O = 4\ CO_2 + 3\ H_2O.$$

The molecule of β-oxybutyric acid contains four carbon atoms, that of diacetic acid four, while that of acetone contains but three. The hydrogen content is eight, six and six in order. The difference between the two acid bodies lies in the absence of two hydrogen atoms in the group CHOH. As would be expected the greater toxicity of the β-oxybutyric acid is the result of the presence of the two extra hydrogen atoms that it contains. It does not seem probable that the liver would waste much effort in reducing diacetic acid to acetone:

$$\underset{\text{diacetic acid}}{CH_3–CO–CH_2–COOH} = \underset{\text{acetone}}{CH_3–CO–CH_3} + CO_2.$$

On the contrary, the following chemical equation would seem much more probable:

$$CH_3–CO–CH_2–COOH + 8O = 4CO_2 + 3H_2O.$$

Most specimens of feces from subjects showing acetone in the urine have a marked odor of this chemical and also show an abundance of bacteria and molds that are known to produce acetone freely in their growth. Careful study of the stools in diabetes leads to the conclusion that the source of β-oxybutyric acid, diacetic acid and acetone is not the metabolic activity of any human organ, but, on the contrary, that they are the direct result of a chronic intestinal putrefactive process.

These three poisons have a strongly selective action upon the islands of Langerhans of the pancreas and the tissue in their immediate neighborhood. From the observation of individuals suffering from diabetes mellitus, acetonemia and kindred diseases the conclusion seems logical that acetone and diacetic acid were formed within the intestinal canal for years before the glycosuria developed; that the pancreas never felt the effects of these toxins until the liver became incapable of guarding the general body parenchyma; and that, through inflammation or destruction of the tissue of the islands of Langerhans the production of pancreatic sucrase was finally so reduced as to render the individual unable to utilize his own glycogen. This, of course, in diabetes of the pancreatic type.

In most sufferers from diabetic conditions it is a comparatively easy matter to check the production of diacetic acid and acetone in the intestines for a time at least, and the improvement shown by these patients is often remarkable. The reduction of the amount of sugar excreted, however, is a different problem. Unfortunately no one has as yet been able to obtain pancreatic sucrase either synthetically or by extraction. Until this is done, our efforts to make diabetic patients utilize the glycogen that every day is returned to nature, a waste product of faulty metabolism, are not likely to meet with much success. To some of these individuals, however, a ray of hope may be extended. There is the possibility that some pancreatic tissue, handicapped by toxemia or by surrounding inflammatory disturbances, may, when its burden is lifted, regain its function. In this instance sucrase

may be elaborated in such amounts as to enable the individual to assimilate carbohydrates in sufficient quantity to sustain nutrition.

In consequence, the amount of sugar found in the urine is not of so great importance is are the strength of the putrefactive indices. These individuals are better nourished and have much greater strength and endurance if considerable amounts of carbohydrates are allowed. When a wound or an abrasion becomes accidentally infected in the course of the disease, a strict starch and sugar-free diet may be put in force. It has often appeared from the study of surgical lesions in diabetes, that too strict a diet had so reduced the resistance of the patient as to court rather than to prevent infection.

This type of putrefaction often precedes severe degenerative processes in the nervous system. Individuals suffering from various psychoses, epilepsy and retinitis pigmentosa, often show this type in a marked degree.

The feces, when acetone alone is present, may be either scybalous, soft or liquid. They are usually dark in color' varying from brown to blackish brown. Mucus is usually normal in amount, but at times may be abundant. The odor is aromatic, often intensely so, and in some specimens vinegar-like. The reaction to litmus may be acid or alkaline, and the blue paper moistened with water and suspended in the containing vessel will often show the presence of volatile acid.

Microscopically, when stained with dilute Lugol's solution, free starch is generally present in considerable amounts, especially if potatoes are a staple article of diet. Striated muscle fasciculi are uniformly absent.

Gram stained preparations show an abundance of large bacilli, measuring six by one microns or thereabout, with rounded ends, capsulated and growing singly, never in chains, always positive unless degeneration has taken place, and never unevenly stained nor granular. Spore formation has never been noted in feces. The next most abundant positive micro-organism is the bacillus bifidus—3 to 5 by 0.2 to 0.4 microns, irregular in form, unevenly stained at times, sometimes bifurcated or of

headlet form. This bacillus bifidus is always found when acetic acid is present in the intestinal contents. Unless the cell body is undergoing degeneration it is always Gram positive, although some bacilli may have negative sections, giving them a mottled appearance. Other bacteria, positive to this stain, such as the capsulatus and the clostridia may be present, but never in great numbers, nor of luxuriant growth. Among the Gram negative forms, the bacillus coli will be found in abundance in the milder types, less and less numerous as the severity of the putrefactive process increases, finally being absent in the most severe grades.

In preparations stained with thionine blue, outside of a few hyphae and conidia, little of moment will be noted.

In the thick films when strong Lugol's solution is used as stain, a few organisms positive to iodine may be found.

When in addition to acetone, diacetic and β-oxybutyric acid are present, the evidences of disease in the stools are much more marked.

Macroscopically they may still be scybalous, soft or liquid, but mucus, either membranous, glairy or blood stained is generally a prominent feature. Unless there is a marked ammoniacal putrefaction present as a complicating condition, the feces are extremely acid and cause considerable burning in their passage through the rectum. The odor of these stools stands alone as the most disgusting encountered in the laboratory. It may be described as alcoholic, aromatic, butyric, acetic, musty, stinking and sickening, and while examining these stools one wonders how any patient could pass such feces without having his attention very forcibly called to the condition of his intestinal canal.

Examined with dilute Lugol's solution, free starch is usually very abundant, clostridia and iodine positive rods of large size numerous and round and oval yeast cells plentiful and of very luxuriant growth. Striated muscle fasciculi may be present if the patient's health has been much affected, if salted meat has been eaten or if cathartics are habitually taken.

The Gram positive fields are especially rich and

will contain clostridia, large round-end rods, granularly stained fusiforms, B. bifidi and B. Welchii in great numbers. The B. putrificus is not regularly present. The negative field is also very rich in bacteria. Large, medium and small rods will be abundant and large hyphae-like bodies often containing chlamydospores, or transparent spores occurring singly or in masses will be found abundantly. The colon type will be absent or only sparingly represented.

It is in the smears stained with strong Lugol's solution, however, that the most characteristic pictures of this diacetic-β-oxy-butyric putrefaction will be found. The richness of these fields in iodine positive fungi of various form is remarkable. Large deeply stained rods often bearing transparent subpolar spores and lightly colored conidia with indistinct outline, are very abundant. The spores often occur singly or in masses and in either instance their capsules take a delicate purple color. Clostridia are numerous, usually sporulating and of luxuriant growth. Masses of a fusiform bacillus will be found, staining delicately with iodine, growing in chains and containing small transparent spores. These chains are often laid side by side, giving the effect of several strings of sausages arranged in parallel lines and in close contact. Smaller bacilli staining delicately and sectionally are numerous. Granular starchy matter and much bacterial detritus staining a light blue will be abundant.

The yeasts, more especially those taking the round form, are very abundant in all microscopical preparations. They very generally contain deeply stained, iodine positive granules.

The carbol-thionine stain may be used to check up the general microscopical findings and in the search for the micrococci of suppuration that may shed some light upon the severity of the local lesion in the intestinal canal.

The biochemical process going on in the intestinal canal among the micro-organisms gaining a parasitic livelihood at the expense of their host presents a very interesting problem. The sugars and free starches are first hydrolysed by the hyphomycetes (yeasts and molds) partly into acids of the lower carboxyl series and partly into alcohols. The

latter are still further attacked by a class of mirco-organisms with the production of acetic acid and its near chemical neighbors. At this point in the process, members of the bifidus group change these acids into ketones, leaving behind those they are unable to hydrolyse. A part of these products of bacterial metabolism is discharged with the feces. Of the part absorbed, the ketones are excreted by the kidneys and the lungs, while the acids are oxidized in the liver. If the latter organ is unequal to the task imposed upon it, while some of the acids may be oxidized by the cells of the general body parenchyma, the major portion leaves the circulation through the kidneys.

The urine in this type of putrefaction will show a marked reaction for acetone, diacetic acid or β-oxybutyric acid, either singly or in combination. High specific gravities are the rule, and the reaction is usually strongly acid. If the pancreas has been attacked sugar is usually present. Many of the urines will contain albumen and casts and these often disappear if improvement is gained through treatment.

The amount of damage this type of putrefaction does to mankind is remarkable. Its onset is insidious and often its presence is never suspected until symptoms arising from the disturbance of the functions of some specialized organ leads to an investigation of the patient's general condition. Unfortunately this usually comes too late. It is common among people who gain their living through severe mental labor, or who are exposed to great emotional strains. Certain families and also certain races are especially susceptible to its ravages. Among individuals who are the misguided victims of vegetarianism, and who find a great financial saving in a meat-free diet, this disease is very prevalent and fatal.

CHAPTER VIII

THE OXALIC TYPE OF INTESTINAL PUTREFACTION

There are certain sufferers from enterocolitis who present a most peculiar history. The primary stage usually escapes notice, although it may be severe enough to confine the patient to bed. In the latter case, intense muscular pains requiring anodynes for their relief, weakness and disordered defecation, with or without a discharge of mucus per anum, are prominent symptoms. The primary stage may last from a few days to a week, or even longer and the importance of the attack, as the beginning of a long siege of ill health, is not realized at the time.

The secondary stage is ushered in with a period of fair bodily comfort of some length, but the patient notices that his strength and endurance are less than they were before the primary sickness. Muscular pains, either constant or intermittent, begin to increase until the patient is rarely free from suffering. This condition progresses until his nights are spent in futile efforts to find a comfortable position in which he may rest without pain and his days in regaining, through massage and exercise, his muscular functions. The muscles are not swollen but are at times extremely tender and through disuse become flaccid. This flaccidity leads to the development of acquired enteroptosis, flat foot and other myastheniae, which add their burdens to those the patient already carries.

The abdominal symptoms are usually of little moment, but in many of the cases, the intense burning and tenesmus at stool and immediately afterward will upset the most calm and phlegmatic disposition. These people resort to cathartics very early in the disease as they find that a series of diarrhoeal discharges have a very beneficial effect upon the

severity of the muscular pains. This free catharsis, so far as the intestinal condition is concerned, only adds fuel to the flame. The mucus is increased remarkably in amount, the stools become more acrid in their nature and a very bad matter is made much worse.

There are many infallible remedies for this so-called rheumatism in the market, each accompanied by a guarantee of sure cure. These are tried in turn, the drastic cathartic effect of each adding its quota of damage. Various irregular practitioners will probably have tried their methods upon the case until, thoroughly steeped in their persuasive conversation, the patient cannot tell whether he is suffering from a displacement of the tenth rib pressing on a nerve, or an exaggerated condition of sin and fear. There is one fact very patent to him, however, and that is that he is growing more inefficient day by day, while to outward appearance he is enjoying at least fair health.

In another class of patients the production of oxalic acid and oxalates is intermittent or cyclical. Sudden attacks of very acute pain, referred to the liver, kidneys or muscles, occur periodically. Under the rigid diet the patient enforces in the few weeks following his illness, the periods of remission are passed in comparative comfort. There will be very few days, however, that the signs of this type of putrefaction cannot be found in the urine and feces. As time effaces the memory of the former attack and the patient uses less discretion in the selection of his food, the train is laid for another explosion. This course may continue for many years and as the importance of the lesion dependent upon foreign microscopic crystalline structures in the body parenchyma does not make a great impression upon the average patient, treatment is very seldom continued. Finally, a trace of albumen found by some insurance examiner or a hematuria brings the patient to the realization of the seriousness of his illness.

His appetite may be enormous, with an especial craving for desserts of various kinds. He may proudly tell you, on the other hand, that animal food could not account for his condition, as he had carefully avoided it for some years.

These are the most important symptoms in the picture of this disease. Other organs may begin to show disturbances of function without demonstrable organic lesions.

The feces may be either formed, soft or liquid, may or may not contain much mucus and are usually of a musty odor. Chemically they may be acid or alkaline, more generally the latter, and show a pronounced reaction with Ehrlich's aldehyde. Microscopically, free starch may be present in the more severe grades and absent in the lighter types. Muscle fasciculi are generally absent. Among the crystalline structures those of calcium oxalate may be very abundant, but fatty acid crystals will be very few in number. In Gram stained preparations among the positive micro-organisms many rods of large size with square ends having a positive line across each end and one along the side, with a Gram negative cell body will be found in abundance. Also cuboid spores, single-celled, having a length twice their diameter, capsulated and occurring singly will be abundant. A few bacillus of bacterium Welchii and also a few bacilli bifidi will be noted. The Gram negative field in the milder grades will show an abundance of bacillus communis coli.

Urinalysis will show no evidence of renal disease in the mild cases; in the more severe cases, however, an albumenuria, which is usually intermittent will often be noted. The indices of intestinal putrefactions of other types will be very weak or lacking, but the amount of oxalate of lime, found in crystalline form in the sediment of the urine will be remarkable.

In the tertiary stage casts, either hyaline, granular or bloody may be abundant.

If the feces are sown on glucose agar, at the end of several days at room temperature, a fuzzy, velvety growth will appear. Stained specimens of this growth will regularly show the presence of monilia.

The tertiary stage may begin without violent symptoms. The lesions of some organ important to metabolism and excretion at the onset may not attract attention. On the other hand, this stage may begin with such violence as to

lead to the diagnosis of an acute lesion. In this connection the estimate that ninety per cent of cases with a diagnosis of acute nephritis are found on microscopical examination to be chronic is especially impressive.

As the disease advances into the tertiary stage the symptoms peculiar to the secondary stage are gradually lost as the more alarming syndrome develops. The patient sinks gradually and dies with a diagnosis of nephritis, myocarditis or some other condition degenerative in character, affecting some vital organ.

An enterocolitis with this type of putrefaction may be called the great moniliasis of the temperate zone. Although many authors have described it as a condition of little moment, still the presence of oxalate of lime crystals in the tissues and more especially in the kidneys is capable of causing lesions from which the patient may ultimately be incapacitated or in many cases meet his death.

CHAPTER IX

THE OLEIC TYPE OF INTESTINAL PUTREFACTION

There are certain sufferers from enterocolitis who show a decided intolerance of fats and oils, combined with severe disturbances in the nervous system. This latter may vary in severity from pyschoses of very alarming character, often requiring institutional treatment, to mild neuralgias or neuroses affecting various organs.

The onset of the trouble may be either sudden or gradual, usually sudden, and, as in many other intestinal infections, the importance of the primary sickness may be overlooked. Rarely, however, will patients be encountered who will not give a history of an acute illness of greater or less severity, from which they date the beginning of their troubles.

The primary stage may begin with a most intense prostration of both mental and bodily functions; and the attack is often so violent as to cause acute mania, suddenly developing melancholia, insanity or in some cases death.

The secondary stage is usually prolonged, lasting in some instances several decades. The psychoses that have developed in the primary stage may continue or may disappear spontaneously. Often new disturbances of cerebral functions make their appearance one after another, until the patient is unable to be cared for outisde of some hospital for the insane.

Another class of individuals develop a special clinical picture, in which a progressive bodily weakness, mild paralyses and severe intestinal symptoms play an important part. Many of these patients show an extreme sensitiveness of the cutaneous surface to sunlight.

The intestinal symptoms in the primary and secondary stages appear of so little moment, in comparison with others of more alarming nature, that they are overlooked or ignored.

Three or four stools a day are not generally regarded as remarkable by the average attendant. These stools are at times accompanied by considerable burning or tenesmus, but the patient is generally in no condition mentally to take much notice of the fact. In the secondary stage, while light yellow diarrhoeal stools are often found, there may be a severe grade of constipation. A very marked increase in the amount of the feces is always present. Mucus may or may not be present, and, if erosions or ulcerations arise as a complication, blood in greater or less amount may be passed.

The blood begins to show evidence of toxic damage very early in the disease, the anemia being characterized by a rapid destruction of erythrocytes. It is rare to find any marked leucocytosis, unless the special point of attack of the intoxication is the spleen or lymphatic system.

From the physical examination of the patient little light will be thrown upon the conditon. The liver may be smaller than is normal, or the colon may show the signs of atonic or spastic colitis. In most cases the abdominal examination will be negative. An examination of the feces, however, will reveal the cause of the trouble.

Macroscopically, the stools may be soft, hard or of creamy consistency; the color light yellow, yellowish gray, brownish gray or clay colored, the brightness of their color varying with the amount of fat crystals that they may contain; mucus is generally present in considerable amounts, although it may be absent. The reaction of these stools may be acid, neutral or alkaline. They are, as a rule, however, acid to litmus. Emulsified with dilute Lugol's solution and examined under the microscope the number of fat crystals present is usually remarkable. These may appear in the form of needles, crystals or in the form of sheaves. The size of these crystals will vary according to the kind of fat most abundant in the food. As these crystals are examined, one is impressed with the great loss of fat that is going on in the intestine. Soap crystals may also be abundant. Free starch and muscle fasciculi are usually absent.

In Gram stained preparations, among the positive micro-

organisms, very large dumbbell diplococci with capsules 5 x 2.25 microns in dimensions will occupy a very prominent place. These often grow in the form of large streptococci composed of the above named dumbbell diplococci. In some specimens the edge of the micro-organism has a wavy outline, putting one strongly in mind of the form taken by a section of inflated colon. In some specimens of feces this micro-organism may be present in almost pure culture. Various other forms such as the biscuit shaped bacterium Welchii, the smaller sausage-shaped rods, the bacillus bifidi, and a few clostridia are generally present in moderate numbers. Unless the feces have been retained in the colon until bacterial autolysis has taken place, the dumbbell diplococci will be found abundant and of luxuriant growth.

Among the Gram negative forms very large hyphae 10 to 12 microns in length by 2 microns in width, which often contain large polar or sub-polar transparent spores, will be noted. These rods at times stain positively in areas. Many fine rods closely resembling the bacillus liquefaciens ilei will be abundant, especially in soft or liquid stools. The bacillus coli, when the disease is of mild nature, is uniformly present. In the more severe conditions, however, it is generally absent.

In preparations stained with carbolic thionine the dumbbells and large Gram negative bacilli will be stained a dark blue in distinction to the other micro-organisms, which appear violet in tint. Often other large conidia-like bodies, staining diffusely, with transparent areas will be found in these specimens.

Smears stained with strong Lugol's solution are usually negative. Occasionally, the large transparent spores, mentioned above, will be shown very plainly, often in masses of seven to ten. The field may be mixed, slightly or absolutely positive, but never negative.

Feces sown on glucose agar media at the end of three days show a slimy, flat growth, spreading over the surface. This growth is white with a yellow tinge with even border and is slightly raised above the surface of the agar. In Gram stained preparations of this growth a few irregularly shaped hyphae

will be noted but no well developed mycelia. The conidia will be fusiformed, capsulated and if sufficiently decolorized with alcohol or nitric acid alcohol will show characteristic Gram positive polar dots. This is a mold of the genus torula.

Much work remains to be done upon the molds that gain their living in the intestinal contents. It is probable that many disagreeable, if not dangerous or fatal disturbances which are occasionally encountered in clinical work may be attributed to the toxins and enzymes that they produce in their metabolism.

Judging from the amount of free oleic, palmitic, stearic and other heavy fatty acids that are present in the feces in this condition, there must be an enzyme of remarkable activity produced in the intestinal lumen. Fat needles are often found in feces of individuals who do not show the same severe nervous symptoms that have been described. In this case, however, the fecal fields do not show the presence of the oleic type of putrefaction and when this latter condition pertains, some pancreatic disease of a functional or organic nature should be suspected.

While admitting that oleic acid and its near neighbors in the fatty acid series may be toxic, when used subcutaneously, it is difficult to believe that the great damage to the nervous system, encountered in this type of putrefaction, can be accounted for on the theory of oleic acid absorption from the intestines. It seems much better to explain this group of symptoms upon the basis of a lipase intoxication arising from the metabolic activity of micro-organisms in the intestinal contents. Sufferers from this type of enterocolitis, when the excessive fat splitting process is checked, show a most remarkable improvement in their mental and nervous condition.

The urine is usually negative both microscopically and chemically.

The tertiary stage in this type of putrefaction is characterized by marked degenerative changes in the nervous system. The brain is the usual seat of this attack and the more severe pyschoses when present render it absolutely necessary that the patient pass his days in some institution.

CHAPTER X

THE AMMONIACAL TYPE OF INTESTINAL PUTREFACTION

There is a type of enterocolitis of a very severe grade, in which ammonium is produced in great quantities in the intestinal canal, accompanied by a persistent diarrhoea, great physical weakness and a very high grade of anemia.

In the primary stage great and sudden prostration, diagnosed as neurasthenia, with rarely a rise in temperature, a weak and rapid pulse, vomiting, abdominal pain and tenderness often so severe as to suggest an acute peritonitis, are all prominent symptoms. Commonly diarrhoeal stools are present, often containing mucus and blood or bloody mucus. In some instances, however, persistent constipation will be found. The discharges per anum may contain very little, if any fecal matter and may consist solely of mucus or mucopus. The illness may be so severe as to cause death or convalescence may be prolonged for months or even years.

The secondary stage may last for several decades and patients passing through this stage are usually great sufferers. There is rarely impairment of the mental faculties, but neuralgia of great severity is a very frequent symptom. Attacks of herpes zoster are very common, often followed by postperpetic neuralgias, which make the patient's life one of misery. The circulatory systems show a marked weakness of the musculature of the heart and blood vessels, but rarely is there any increase in blood pressure or an arteriosclerosis.

In the urinary system secondary infections are generally the rule, leading to pyelitis and cystitis with ammoniacal urine. The precipitation of the earthy phosphates of the urine is very common and in patients who have suffered for several years from this type, one or more phosphatic calculi are usually present, in the kidneys, ureter or bladder.

The liver may be enlarged, its lower border swollen, rounded and soft. The gall bladder and gall ducts may also be the seat of calculus formation. The colon may be palpable, prolapsed, distended, crepitant or knotty from the presence of scybala. Intense pain may be referred to its location with burning of greater or less degree. Under X-ray examination, portions may be found showing constrictions, which may be either organic or spastic in origin.

The urine is light in color, cloudy from the presence of pus or earthy phosphates and intensely ammoniacal in odor. The reaction is uniformly alkaline. The specific gravity is as a rule so low as to lead to a suspicion of an incipient interstitial nephritis. As recovery takes place, however, the specific gravity usually rises to normal. Albumen may be present in greater or less amount. An indicanuria of high or low grade may be present, but is not, in simple cases, prominently so. Diacetic acid may or may not be present and also skatol.

Upon microscopical examination, the great number of crystals of ammonium magnesium phosphate, together with the abundance of earthy phosphates, will be remarkable. The presence of casts of various types gives the condition a place in the tertiary stage and organic elements are rarely found in the secondary stage.

The feces may be liquid, watery, formed or scybalous. The color may vary from yellowish gray in diarrhoeal to dark brown in scybalous stools. Mucus may or may not be extremely abundant. The intense ammoniacal odor of these stools is remarkable. The stools are always strongly alkaline. Litmus paper wet with water and held over the mouth of the container will be turned blue.

Microscopically, with dilute Lugol's solution, free starch is absent, muscular fasciculi are absent, a few fat needles may be noted, yeast cells, usually of the round variety, will be abundant, while of the crystalline bodies, the number of triple phosphates will be a cause for remark. Many soap crystals will also be found.

In the Gram stained field, among the positive forms, the

presence of streptococci will be especially noticeable. Many of the capsulatus type will be found, as these grow luxuriantly in alkaline feces. If ulceration is present, cocci and staphylococci will be abundant, their number varying with the nature and extent of the suppurative process. Long filamentous forms, apparently belonging to the B. putrificus group, are very abundant. Occasionally, clostridia will be present, the alkaline intestinal contents furnishing a very favorable medium for the growth of acid producing bacteria.

In the Gram negative field, many large rod-like microorganisms will usually be observed. These, apparently, are the hyphae of molds and may have sections taking the positive stain with more or less intensity.

Various negative forms, such as the B. liquefaciens ilei may be present. The colon bacilli in the more severe types are absent. In their absence or presence, and if present their number, we find a very useful method of estimating the severity of the intestinal condition.

In the preparations stained with carbol thionine the microorganisms are generally stained blue. The capsulatus types usually take the violet tint which is characteristic in specimens from the various types of putrefaction.

With the specimens stained with strong Lugol's solution, free starch detritus is very regularly present. Clostridia may or may not be present. Often the micro-organisms in the immediate neighborhood of the free starch granules take the iodine stain in the less serious types of this putrefaction.

The microscopical field varies from absolutely positive to mixed, concomitantly with the severity of the enteric catarrh.

There are many patients in whom the severity of this intestinal condition is masked through the coexistence of a putrefaction of an acid type. So long as the production of acids and ammonia is about evenly balanced, these patients enjoy fair health and are able to perform their daily tasks in comparative comfort. At times, either through changes in the character of their diets, or, perhaps, through the acquisition of new and more active strains of either acid or alkali-producing bacteria, the balance is upset, and they suffer

attacks attributable to either one effect or the other. The character of this condition can be easily discovered through the methods of examination set forth in the previous chapters.

Marked organic lesions do not generally mark the tertiary stage of this type of enterocolitis. The patients are gradually worn down by attack after attack of an acute nature until they lapse into a condition best described by the term, general debility. A rapidly advancing pernicious anemia or some intercurrent disease, usually ends their life peacefully and with little suffering.

CHAPTER XI

THE URIC ACID TYPE OF INTESTINAL PUTREFACTION

There are certain patients, the victims of mild grades of enterocolitis, in whom there seems to be a chronic deficiency of oxidizing power. This may be a matter of heredity, acquired as the result of sedentary habits combined with the habitual ingestion of great quantities of highly nutritious food, or the result of exhaustion of the oxidizing function through the consumption of alcohols, ethers, aldehydes, acids and aromatic bodies found regularly in the products of the vineyard and of the still. In consequence, a very slight increase in intestinal putrefaction upsets their metabolic equilibrium, while any sudden increase in the production of acids in the digestive canal brings on an attack of gout of a severity varying concomitantly with the enteric condition.

The important point in these cases, however, is that there is an intestinal putrefaction of some type present, not only at the time of the paroxysm, but also during the time when gouty symptoms are absent. If this can be checked and a normal condition of the intestinal mucosa brought about, the patient will experience no further difficulty. The intestinal condition may be diagnosed through the methods laid down in this work.

CHAPTER XII

MALARIA, SYPHILIS AND TUBERCULOSIS

In every case of enterocolitis, no matter what the type of putrefaction may be, an investigation of the possibility of a malarial, syphilitic or tubercular infection, singly or in combination, should always be conducted.

Malaria is one of the most common diseases of civilization. The malarial chill occurring every two or three days has, in New England, become a rarity. In its place we find the chronic, deep-seated infections of long standing in which a diagnosis is extremely difficult from an examination of the blood unless the parasites are in the swarming stage. Often two or more types of malarial parasites may be found in the same person. The spread of malaria through the New England states may be laid at the door of immigration. The subtertian type is the most prevalent.

In order to get a correct clinical picture of these malarial conditions we must regard them as a chronic parasitic disease of the liver, spleen, pancreas, bone marrow and other organs, and not of the red blood cells alone. The time during which these parasites lie dormant in these organs may be passed in comparative comfort so far as the patient is concerned. At certain seasons of the year when the blood stream is invaded by the sexual forms of the plasmodium, or their growth becomes more active, various intestinal symptoms are prominent. These may vary from distress, burning, epigastric unrest, diarrhoea or constipation, to pain in the right side of the abdomen so severe as to simulate appendicitis.

There is usually no rise in temperature at the time of these attacks. Upon physical examination, the liver is usually found swollen and palpable and often tender. An examination of the blood will show a mild leucocytosis.

Better results will be obtained in the search for plasmodia

if the blood is examined after the method of Manson. A glass slide and cover glass are first carefully cleaned with alcohol. It is especially important that these should be absolutely free of all grease, before spreading the blood. The lobe of the ear is next punctured with a small lancet or a three-cornered needle, a very small drop of blood placed in the centre of the cover glass, great care being taken that the glass does not touch the skin. The slide is then breathed upon and the cover glass placed blood downward upon it. This process must be done quickly in order that the drop of blood may not dry. When this is properly done, three zones will be noted in the preparation. The central zone should be absolutely transparent and colorless, the middle zone should have a delicate pinkish yellow tint and the outer zone should be blood red. When examined with a twelfth oil immersion lens, in the central or colorless zone the blood will show scattered erythrocytes, resting flat between the two glass surfaces. In the light colored zone the red cells will be overlapping or rouleated with clear spaces between. The red area will consist of closely packed red cells with here and there a leucocyte. The central transparent area should be carefully searched for plasmodium, either within or upon the red cells. The second area should be examined for active parasites and for micro-and macrogametes. These will not be found in the transparent area as the pressure of the cover glass upon the glass slide prevents them from freeing themselves from the red cells. These gametes at room temperature are generally extremely active.

Stained blood smears are usually of less value than fresh preparations.

The malarial infections met with may be classified as follows:

The Quartian Type, Plasmodium malariae.
The Tertian Type, Plasmodium vivax.
The Pigmented Quotidian Type, Laverania praecox.
The Unpigmented Quotidian Type, Laverania immaculata.
The Subtertian Type, Laverania malariae.

Any organ of the body may become sensitized to the malarial protein. The symptoms that the patients show will depend upon which tissues have acquired the power of digesting the protein of the plasmodia. The blood cells may have acquired this property and the remainder of the body parenchyma, not having the same power, may remain intact. In this case a true picture of pernicious anemia will be given. So, also, the epithelium of the mucous glands and follicles of the intestinal canal may become sensitized to this poison and the other organs of the body remain unsensitized. In this case the patient will give a history of subacute attacks or exacerbation of a low grade catarrhal process, limited to the intestinal canal. Having confirmed the diagnosis by a microscopical examination of the blood, it is extremely interesting to follow the course of the intestinal symptoms under antimalarial treatment. Most of these cases are made very much worse by quinine, at least during the first week of treatment. The reason for these untoward symptoms arises from the fact that the tissues of the intestinal canal during this period are called upon to digest the malarial protein arising from the death of the plasmodia through the toxic action of quinine, in far greater amount than before. In about two weeks after the beginning of anti-malarial treatment, it will be universally found that larger doses than those used in the beginning may be used without disagreeable symptoms.

The fact must be borne in mind that in many of these individuals the power of digesting particulate malarial protein bodies has been exhausted. In other words, that the patient has developed a condition often termed anti-anaphylaxis. The blood in this condition will show a marked leucopenia. The scarcity of the white cells and also their lack of vitality will be noted in the fresh blood preparations if the warm stage is used with the microscope. In normal blood preparations the living leucocytes should never take the spherical form at body temperature. The normal leucocyte is always angular or amoeboid in form. Therefore, so far as the leucocytes go, the number of these cells may be used as a guide to the resistance of our patients against the infection.

The cover glass had better be ringed with vaseline to prevent evaporation in the blood film and consequent crenation of the red cells that might simulate plasmodia attached to the cell surface. An abundance of crenated erythrocytes indicates a deficient vitality of the red cells. If any of these cells have a to-and-fro motion, we may assume that a malarial parasite is attached to it. The study of the minute bodies observed in the plasma of the fresh preparation will lead the examiner to conclude that possibly a sexual cycle takes place in the blood stream, as well as in the mosquito.

The organisms are often pear-shaped, motile and appear to attach themselves to the red cells by their smaller extremity. Often small poikilocytes and microcytes will be seen in motion, traveling across the field, towed by these pear-shaped micro-organisms. When these larger bodies are free-swimming they take the form of a dumbbell. Their motion is very active. Their bodies are highly refractile and the cytoplasm shows evidence of intracellular circulation. Apparently this body eventually divides the two pear-shaped bodies.

Crescents will also be noted attached to withered or crenated erythrocytes. This form is not so active as the pear-shaped and dumbbell forms. They are rather hard to distinguish under the microscope as the light transmitted through the red cells gives their bodies the same tint as the colored corpuscles of the blood.

The course of malarial infection may be divided into four stages. First, the stage of invasion; second, the stage beginning with the establishment of sensitization, which may be called the anaphylactic; third, the stage in which the growth of the plasmodia and the parenteral digestion of the malarial protein is evenly balanced; fourth, the stage in which the power for protein digestion has been exhausted and the patient enters a condition usually described as anti-anaphylaxis.

The primary stage or the stage of invasion is devoid of serious symptoms. The patient may have certain prodomata, such as mild headache, slight loss of appetite, weakness and lack of endurance, but does not consider himself ill.

The second stage usually begins with a pronounced chill,

followed by a rise in temperature, which in turn is followed by profuse perspiration. This corresponds to the time when the patient's sera, leucocytes and tissue cells have acquired the power of digesting the particulate protein of the malarial plasmodia. This stage may last from one day to two weeks, depending upon the strength of the special strain of malarial plasmodia that are invading the body.

In the third stage, very few symptoms will be shown outside of a weakness and loss of strength and endurance.

In the fourth stage, where the resistance against the attack of the plasmodia has been completely exhausted, the patient's life becomes one of great misery. In this stage, the symptoms may be referred to any of the specialized organs, either singly or in groups embracing two or more organs. The central nervous system is very apt to be the seat of attack and certain comatose, paralytic or neuralgic conditions may be encountered. In the tropics these conditions are frequent and fatal, but in the temperate zone, while not so severe, they are far more common than is realized. Conditions diagnosed as serous apoplexy, sudden cerebral congestions, severe neuralgias and attacks of herpes zoster, with or without postherpetic neuralgias, may be caused by a chronic malarial infection in the fourth stage. The circulatory system may be the point of attack and various functional cardiac disorders, often accompanied by a rise in blood pressure, irregular heart action and precordial pain will be noted. If the liver is the point of attack, pain referred to the right side of the abdomen, jaundice, swelling of the liver, attacks with fever, often described in the tropical countries as bilious remittent fever, will occur. The pancreas may also be the seat of attack with glycosuria, pain and fatty stools. The kidneys may also be attacked and syndomata resembling the black water fever of the tropics, but much less severe, may be observed. Intestinal symptoms will often be prominent. These usually show a certain periodicity and may be accompanied by constipation or diarrhoea, a great increase in the amount of flatus and an abundance of mucus in the stools. Pain is apt to be a prominent symptom and may be referred to any por-

tion of the abdomen. So severe may these pains be as to simulate appendicitis, biliary colic, and many of the operations undertaken for the relief of the two latter conditions in which no lesion of the appendix or the gall bladder are found may be laid at the door of this disease.

A few words upon the life history of the micro-organism of subtertian malaria may be of interest. Let us begin with the mosquito of the variety anopheles, in which the salivary glands are charged with the microzoon of this disease. Upon biting a human being the germ enters the lymph channels or the capillaries. In a longer or shorter time, depending upon the number of organisms introduced, the body fluids will contain billions of plasmodia. This may or may not be followed by a sensitization of the general parenchyma or of certain organs or groups of organs. The more general the sensitization, the less liable will the patient be to a chronic malarial infection. The entire crop of plasmodia may be destroyed at the time of the first chill. On the other hand, several chills, each one of less severity than its predecessor, may lead on into the third stage of chronic malaria. If this occurs, the plasmodia penetrate deeper and deeper into the body tissues and are found no longer in the peripheral circulation, finding their abode in the liver, spleen, brain or bone marrow. At certain periods these plasmodia set free a host of micro- and macrogametes, but between times the blood, so far as micro-organisms are concerned, is free of malarial forms. The time of swarming usually corresponds with the period of malarial symptoms in the individual. If blood containing these forms is eaten by the anopheles, the mosquito becomes infected and can transmit the disease. On the other hand, two cycles may take place within the body, an asexual and in all probability a sexual cycle. The latter type of propagation seems very probable, in view of the fact that asexual propagations among all protozoa, without the introduction of new individuals in the chain, eventually result in the degeneration of that individual colony. Many of the blood pictures seen in the study of chronic malaria lead to the prediction that this will some day be proven.

It seems also probable that malaria may be transmitted in other ways than through the agency of anopheles. More especially when we consider the minuteness and motility of the micro- and macrogametes of the subtertian plasmodia. Possibly, as in syphilis, this type may be transmitted by direct contact with abraded surfaces, or upon the ovules or microgametes of human beings.

Often no second stage will be noted and the patient will become malarialized without having any of the classic symptoms of an acute malarial infection. This will account for many of the chronic malarias that give no history of any acute attack.

Three types of syphilis will be met with as complications in enterocolitis. The first type includes ulcerations, gummata and other purely local lesions of the intestinal canal. The second type includes syphilitic disease of the liver, pancreas and spleen. The third type includes diseases of the nervous system, acting reflexly upon the intestinal canal and also lesions of the intestinal mucosa, corresponding in their pathological processes to the tertiary lesions found upon the skin. In the first two types the Wasserman reaction is usually positive for the blood. In the third type the cerebrospinal fluid will be positive and the blood negative. In the first type the lesions will be caused by the direct attack of the treponema. In the second, the symptoms will be secondary to syphilitic disease in the organs supplementary to intestinal digestion. In the third, the symptoms will arise from the disturbance of the correlation of the mechanical and chemical processes of digestion, in the first case, and in the second, from sensitization phenomena without the local presence of the micro-organism of the disease.

The lungs should always be carefully examined for tubercular lesions and the possibility of tubercular foci in other organs should always be considered. Primary tuberculosis of the intestinal mucosa, while rare, is always possible as a complication of an intestinal catarrh. If ulceration is present, tubercule bacilli and blood will be present in the feces. A search for tubercule bacilli is always important in the examination of feces.

CHAPTER XIII

THE PROTEIN POISON

Protein sensitization or anaphylaxis occupies a very prominent position in many of the problems of enterocolitis, and the parenteral digestion of foreign protein bodies and the poisonous effect of the split products of this digestion should be kept in mind. These bodies may enter the intestinal canal with the food, in the mucus from the nose and throat or arise as the result of bacterial growth in the intestinal canal, the passages connected therewith or in the mucosa or submucosa that lines them.

In nearly every individual, the phenomena of sensitization against certain proteins can be found. In some the proteins of fish, in others those of eggs, while in others those of certain vegetables and fruits cause disagreeable symptoms or at least are extremely distasteful. Other sufferers from enterocolitis notice that certain months of the year are especially uncomfortable so far as their intestinal condition is concerned, and so regular is the recurrence of these periods that a direct connection with some of the pollens that cause hay fever sufferers so much trouble may be suspected. The desquamations of some of the lower animals may also be a cause of trouble.

In the primary stage of enterocolitis, sensitization phenomena are generally very prominent, often alarming in their intensity and may in certain cases be the cause of sudden death. Most of the attacks of acute indigestion are caused by the so-called protein poison, arising from the digestion of the particulate protein of bacteria or of soluble proteins that have escaped the action of the enzymes normally present in the intestinal canal.

The mucous surfaces of certain individuals seem to be

especially permeable to certain protein bodies. Ulcerated or eroded surfaces and large areas of scar tissue make this absorption especially hard to control.

In the case of the particulate proteins, namely the micro-organisms, this sensitization gives complete protection against the further attack of the invading parasite so long as this immunity lasts. In the case of the soluble proteins, however, the amount absorbed may be great enough to exhaust the sensitization of the cells of the body and bring on the condition termed anti-anaphylaxis. This condition is present in many cases of enterocolitis. Consequently, the success or failure of our efforts to relieve our patients depends upon our ability to find which proteins are harmful and which are well borne.

The symptoms in the primary stage may best be grouped under the term acute-anaphylactic or sensitization shock. These are paralysis of the voluntary muscles, a greatly reduced blood pressure, a spasm of the bronchi and death by suffocation resulting from this spasm. This train of symptoms is present in every sufferer from acute indigestion. The patient falls, struggles for breath, goes into a state of syncope, respiration ceases, and he dies, so to speak, with his heart still beating. If recovery takes place the respiration first returns, the weakness rapidly disappears and the patient recovers in a remarkably short time. The prodroma that are noted in animal experimentation are always well marked, namely the itching of the skin or a disturbance of cutaneous sensation.

CHAPTER XIV

THE TREATMENT OF ENTEROCOLITIS

The therapy of enterocolitis furnish many problems of great interest. Upon the success or failure of our efforts, the future usefulness, comfort and prosperity of our patients will depend. It is well to realize in the beginning the magnitude of the task, to gain the confidence of the patient and to avoid promising too speedy a recovery. Many of the patients improve wonderfully in a few weeks, take false courage and believe that their health has been permanently restored. Such people should be told frankly that their improvement may be temporary and that the fact that it took place does not affect the length and persistence of the therapeutic campaign. The average patient expects great things from the physician and is apt to form a poor opinion of any medicine that does not bite as quickly as the ubiquitous cocktail. Procedures that neither physic nor benumb are beyond his comprehension. Quick results are always expected and it is hard to convince him that the physician cannot supply that very important factor in the treatment of his disease, namely time. It is also difficult to protect your patient from the advice of friends, nurses and irregulars, often to be sure, given with the best intentions, but, from a psychological standpoint, very deleterious.

While realizing that humanity is prone to err and that the physician should be ever ready to forgive any lapses from routine that are confessedly the result of human weakness, still, never allow a patient to argue this question. The matter should be brought at once to the point of obey or find some other physician. The mental process of the patient who has a history and physical examination completed, submits specimens of gastric contents, feces and urine, calls once and is never seen again is hard to explain. This very frequently

happens and always awakens feelings of regret that the time spent in the study of the case has been wasted, and that the laboratory force has been needlessly exposed to danger.

In the *primary stage* of chronic enterocolitis more can be accomplished with a little treatment than in any other known disease. The way is open for direct local therapeusis, but it is absolutely necessary that this treatment should be appropriate for the condition of the mucous membrane and the type of infection.

The type should be diagnosed through analysis of feces and urine, the anatomical features cleared up by a thorough physical examination and a search for any organic lesions carefully made.

The patient had better rest quietly in bed until the acute period is passed and for good measure a few days more, in order that his strength and resisting power may be completely regained. If possible, he should be kept under observation until the urine is negative, so far as the indices of intestinal putrefaction are concerned and the fecal fields Gram-negative. If after a month has elapsed, the chemical and bacteriological findings are still negative, the patient may be discharged as cured. If, on the other hand, at the end of ten days, the chemical and microscopical findings are still positive, the patient may get up and about and gradually resume the duties of life, provided the temperature and pulse are normal and the general condition warrants it.

Our efforts in this stage should be directed toward the accomplishment of four things: First, the restoration of aseptic conditions in the gastrointestinal canal; second, the healing of the catarrh; third, the complete digestion of any foreign protein bodies that have found their way into the mucosa or into the body tissues; fourth, the limitation of the amount of damage that may be done to any organ during the course of this stage.

To accomplish the first result, the administration of intestinal antiseptics is the treatment par excellence. To avoid any irritation of the intestinal mucosa, they should be given in small doses. Certain antiseptics have been found especially

potent against certain types of bacterial invasion and the smallness of the amount required to produce results is often surprising. The amount of the salycilates in the lymph circulating through the ligaments in acute articular rheumatism must be very small and yet the results are remarkable. So also the results that we may get from the use of antiseptics in the intestinal canal vary with what we might term the particularity of those we administer. The antiseptic which in the smallest doses and with the least irritation will acccomplish the greatest good is the one to be preferred. Intestinal antisepsis is still a matter that gives great room for research. The day is not far distant when the enzymes, the keys fitted for unlocking the proteins and toxins of each special type, will be in common use for the benefit of humanity.

In clinical investigations the following antiseptics have been found the most useful in the acute stages:

Sodii Salicylas.
Dose; Gms. 0.064 or 1 grain every two hours.

Hexamethylenamina.
Dose: Gms. 0.064 or 1 grain every two hours.

Oleum Ricini.
Dose: Gms. 2.0 or 30 minims, every four hours.

Acidum Nitricum Dilutum.
Dose: Gms. 0.064 to Gms. 0.128 or Mi to Mii every two hours.

Bismuthi Subnitratis.
Dose: Gms. 0.250 or Gr. iv, every two hours.

Sodii Benzoas.
Dose: Gms. 0.064 or Gr. i, every two hours.

Calomel in divided doses.

The sodii salicylas will be found most useful in the saccharobutyric, acetic and oleic types of putrefaction. The hexamethylenamina in the indolic and oxalic types. The oleum ricini, given in the doses mentioned above, is almost a specific in the oleic type and of great value in the oxalic types of putre-

faction. Bismuthi subnitratis and acidum nitricum dilutum are of especial service in the oxalic types, although the bismuth may be administered with advantage to most sufferers from enterocolitis in this stage. Occasionally, it acts poorly, irritates and seems to encourage rather than check putrefaction. The benzoate of soda, while of little value by itself, is very useful in combination with hexamethylenamina in certain individuals, who do not seem to cleave the latter.

Calomel in small doses deserves a firm place among the most valuable remedies in this stage. The smaller the dose, the better its action. To avoid salivation the usual saline cathartic should not be forgotten.

Drugs that act through the liberation of nascent oxygen in the intestinal canal are not generally a success in the primary stage.

The catarrhal process may best be handled through the diet. The catarrh, however, if the intestinal putrefaction is held in check, usually recovers spontaneously. As a routine treatment the tincture of belladonna in small doses, Gms. 0.064 or Mi, every two or three hours, seems to be of benefit. The acidity of the stomach may be lessened or neutralized with sodium bicarbonate and sodium bromide may be used to check disagreeable reflex disturbances.

Vomiting from reflex irritation of the intestine or of the central nervous system may be so severe as to require the administration of morphine by hypodermic injection for its relief.

Sensitization phenomena may form a very important part in the clinical picture in this stage and are often very severe in character. Sudden deaths from acute indigestion are usually the result of shock resulting from the production of beta-iminazolylethylamin. This may be formed in the intestinal canal and be subsequently absorbed, or from the parenteral digestion of foreign protein bodies, which have found their way through the intestinal mucosa. These foreign proteins may be introduced with the food or be the result of bacterial growth. Death is usually the result of bronchial spasm, the heart still beating after respiration

ceases. The treatment, therefore, should be directed to the relaxation of bronchial spasm and to raising the blood pressure. The first can be best and most quickly accomplished by ether inhalation. If the bronchial spasm has reached the point of respiratory suspension, the hypodermic method may be tried or tracheal insufflation of ether vapor attempted. There are numerous drugs that have a vaso constrictive effect and may be administered in appropriate doses. In the laboratory, barium chloride has been found to be a specific for sensitization shock and in no case has death resulted after an injection of solutions of proper strength from the administration of the protein poisons. Its use in human beings has never been investigated, but if its effect should be found the same as in the lower animals, it would be a great addition to our list of life-saving drugs.

The amount of damage done to the mucosa may be limited by rest in bed and by the avoidance of a diet which is irritating. It will be of the greatest advantage to our patient if the healing process has been carried as far as possible before allowing him to return to his regular duties; especially so, if the disease does not show signs of spontaneous recovery.

Drugs given to check diarrhoea or to produce bowel movements should be used with extreme caution. Cathartics retard the healing process and through hastening the intestinal current cause the chyle and feces in the lower intestine to become too rich in nutritive material and thus encourage putrefaction.

The diet in the primary stage must of necessity be bland and unirritating. Milk, preferably cooked in order that it may be sterile and its proteins slightly hydrolysed, makes, in most cases, an ideal diet. Gruels and broths may be used if the milk is not well borne. Often scraped raw beef on toast can be administered with great benefit in the sacchrobutyric, acetic, oxalic and ammoniacal types.

In the *secondary stage* a large and varied armamentarium is an absolute necessity in the fight. The power of the various organisms with which we will be called upon to do battle, to adjust themselves to new conditions and to overcome

the effects of various chemicals which in the beginning were toxic to them, is well known. In the same manner that the plasmodium of malaria will become quinine fast, so will the intestinal bacteria become salicylate fast or formin fast.

Where mixed infections are present the solution of the problem presents even greater difficulties. The suppression of one fauna allows another to gain the ascendency, perhaps to the detriment of the patient. This fact, however, at times may be turned to our advantage, always bearing in mind that there is not only a substitution but also a re-substitution with which to reckon. Remedies often lose their effect after a few weeks and others must be tried in their place. Finally, either the enterocolitis is healed, considerable improvement is gained or the onset of the tertiary stage destroys all hope of a complete recovery and forces us to efforts to postpone that fatal day when some vital organ will cease to functionate.

Indolic Type.

Enterocolitis accompanied with the indolic type of putrefaction, in the primary stage at least, usually heals with great promptness under the administration of hexamethylenamina in small doses, combined with a meat and toast diet.

In the secondary stage the therapeutic course is more prolonged. In the milder conditions, however, the catarrh usually heals in a few months under the administration of hexamethylenamina, the correction of starchy indigestion and a diet combining high nutritive value and small weight. In a few individuals, however, this course of treatment will have little effect, the indicanuria will persist and the catarrh will still advance. In this case sodium salicylate should next be tried and after a week's treatment omitted for twenty-four hours and the urine tested for indican. Should the urine be still positive for indican, hexamethylenamina and sodium benzoate should next be administered and test again made. Meanwhile the patient must be carefully watched for any signs of intestinal irritation. If these should appear, the use of antiseptic should at once be discontinued. In some individuals it will be found impossible to establish a condition

of asepsis or even partial asepsis in the intestinal canal and we must be content with the antiseptic which seems to agree with the patient best and continue its use off and on, while we devote ourselves to the treatment of the catarrh.

In the treatment of the catarrh the following drugs will be found of great service: The wine of ipecac in small doses, the tincture of belladonna, bicarbonate of soda and bromide of soda. The ipecac apparently increases the secretion of the intestinal mucosa, washes out the mucous glands and turns the current towards the lumen of the intestine. The belladonna, while checking secretion to a certain extent, relaxes the spasmodic condition that so often accompanies these catarrhs and relieves many disagreeable symptoms of reflex nature. Sodium bicarbonate lowers the crest of the acid wave originating in the stomach, helps out the pancreatic juice in its effort to sustain the alkalinity of the intestinal contents and renders less irritating the organic acids that the liver will be called upon to oxidize.

All foci of suppuration should be removed, all bridge work should be consigned to the melting pot, all carious teeth extracted and any pyorrhoea cured or failing in this the teeth removed and a plate fitted.

Fingers should be kept out of the mouth and nose and the teeth should be thoroughly cleaned at least twice daily.

The need of antiseptics will be much lessened if the food is as sterile as possible. This condition can be obtained through care in handling raw foodstuffs and especially, in the case of vegetables, by the most thorough cooking. Until the succus entericus has regained its bacteriolytic activity all uncooked foods or foods that have stood some time after cooking, should be prohibited.

In the secondary stage very little headway can be made in the cure of enterocolitis while excessive putrefactive processes are going on in the intestinal canal. The best plan is first to give the patient a course of intestinal antiseptics and to delay therapeutic measures directed to the cure of the catarrh until the indices of intestinal putrefaction are no longer present in the urine.

The name of antiseptic drugs is legion and a list of them can easily be compiled from any text-book on therapeutics. It will be found, however, that the best place to begin the sterilizing process is at the vermilion border of the lips. In some instances the outposts may be pushed as far as the garden, the market, the ice box, kitchen and eating utensils, with what may seem favorable results. In the fight for asepsis the nose and its sinuses, the posterior nares, the tonsils and the teeth must not be forgotten and these matters had best be taken in hand by men who have a correct understanding of the task at hand.

Saccharobutyric Type.

This can best be treated by means of the salicylate of soda given in small doses as this drug is almost specific in its action. Like all other intestinal antiseptics, however, its long continued administration does not give good results. Its first effect will be to diminish greatly the volume of fecal matter discharged per diem, and as this is generally regarded as an alarming condition by the patient, he had better be prepared beforehand for this result. The first course had better last one week and never longer than two weeks, for if longer continued certain butyric acid producers tend to become salicylate fast.

As the first symptoms of this type arise from the fact that the liver is becoming insufficient for the task imposed upon it, and as this is caused primarily by the excess of butyric acid, either free or in loose combination, in the portal circulation, it is well to administer the alkaline carbonates well diluted with water. The liver seems to be better able to handle these acids if they are combined with sodium or potassium. This fact is shown clinically by a decrease in the sensitiveness of the right hypochondrium and a reduction in the size of the liver. Alkaline mineral waters usually have some cathartic action, therefore their general use is not to be recommended.

It must not be forgotten, however, that our greatest efforts should be directed to the healing of the enterocolitis, the

ravages of which allow the growth of butyric acid-producing bacteria to proceed unchecked in the patient's intestinal canal. Drugs, such as ipecac, belladonna, hydrastis, that enjoy a reputation in the treatment of the catarrh may be prescribed, but little can be accomplished through their use if the patient persists in following a diet which favors the growth of those bacteria that are always present in this type of putrefaction.

Diet alone will always benefit this catarrh in the secondary stage and many of the milder cases can be cured by this means alone. For the first few weeks that the patient is under treatment, the diet should be that ordinarily given in diabetes with strict orders to omit all uncooked vegetables. If there is a ptosis or a tendency to ptosis of the viscera, solids and liquids should not be allowed to be taken within two hours of each other. Milk is not well borne by these patients and should always be forbidden.

The micro-organisms of this type are very generally strict or facultative anaerobes, very difficult to cultivate in the laboratory without special apparatus. The patient, moreover, spends the whole of his life in an atmosphere of oxygen of just the right dilution for therapeutic use. The use of enemeta of atmospheric air often produces marvelous results in these sufferers, more especially if diarrhoea is present. This may best be administered with a bulb syringe, the average amount introduced being six ounces. Two bulbsful usually make this amount in the ordinary Davidson syringe. A special hydro-pneumatic apparatus designed for this procedure was found to have no special advantages over the syringe mentioned above.

Enemeta of water, decinormal salt solution, antiseptics and astringents uniformly seem to do more harm than good.

Abdominal massage, electricity and hydrotherapy have been found of little use.

The correction of ptoses by strapping has been of great service where such procedures are indicated. Experience teaches that the simpler the apparatus can be made the better. After trying the various nicely patterned belts

that have been invented, finally one constructed of a strip of zinc oxid plaster 3½ inches wide, applied across the small of the back, brought forward just about the crests of ilium, crossed midway between the umbilicus and the pubes and secured with a pin, was given the preference. The point of this pin is cut off and a small piece of plaster applied to prevent injury to the clothing.

As the patient progresses toward recovery first one vegetable and then another is restored to the diet and the effect carefully noted. Finally most patients can be allowed a regular table diet. On the other hand, many individuals will be encountered who are unable and always will be unable to indulge in salads or other uncooked vegetables.

The Acetic Type.

The acetic type of intestinal putrefaction in the secondary stage can best be handled through the diet and many will recover promptly through this means alone.

The individuals who have an acetonuria alone, may not consider themselves as departing very far from a normal condition, but when the urine shows repeatedly a reaction for this chemical there are sure to be stormy days ahead. Slightly limiting the amount of starchy foods, the administration of a mild intestinal antiseptic, such as sodium salicylate or hexamethylenamina for a few days usually corrects the trouble.

When diacetic acid, oxybutyric acid or the acetic derivatives are present, the therapeutic problem is one of greater difficulty. The so-called antidiabetic diet will be found of great usefulness as a means of treatment. There is one exception, however, that must be carefully enforced and this is the total abstinence from all raw foods, or from any food that has been allowed to stand long after cooking, such as cold meats, cold vegetables, vegetable salads and foods of like nature. In the severe grades of putrefaction even the drinking water should be first boiled before using and the danger of infectious contact should also be guarded against.

The intestinal antiseptics mentioned before may be tried in

turn. There appears to be no fixed rule governing the administration of the class of remedies. In some, the salicylates may be used to great advantage; in others, hexamethylenamina either alone or in combination with sodium benzoate will cause the urine to become negative; while in others, Fowler's solution or potassium iodide will accomplish the desired results.

The intestinal catarrh should be attacked after the indices of putrefaction have either been brought to zero or very materially lessened in strength. Ipecac, belladonna, bismuth subnitrate or subgallate, dilute nitric acid, the alkaline carbonates may be tried and the ones that seem the most useful in this special case administered.

As in the other forms of putrefaction, cathartics are to be avoided as much as possible, in order that the contents of the lower portion of the intestinal canal may be as deficient in nutritive material as possible. If the patient seems to be worse when constipated castor oil, aloin, cascara sagrada, may be tried with or without podophylin. The rule is, however, unless the patient shows dangerous symptoms or the disease has progressed so far as to be well-nigh hopeless, to fight it out with the aid of enemeta and lubricants.

As in the saccharobutyric type, enemeta of atmospheric air, especially if diarrhoea is present as a complication, are often of great service.

Massage, electricity, hydrotherapy and lavage are of very little benefit. A change of scene and climate is of service as it often removes the patient from a source of infection that impedes his recovery.

The treatment of the tertiary stage is a task for the well trained internist. The means taken to gain time for the patient, of course, varies with the organ or organs that have been affected through the attack of the disease. The principles of treatment laid down above will be found of great use in combination with those set forth in treatises upon diseases of the pancreas, liver, heart, blood vessels, nervous system and other organs of special function.

Oleic Type.

In the primary stage of oleic putrefaction, the patients are generally very ill. In consequence, these patients had better be kept in bed until recovery is well advanced.

Castor oil in small doses frequently repeated is well-nigh a specific in this type of putrefaction. It should be given in half dram doses, once in two hours. It may seem paradoxical that a type of putrefaction where the invading microorganism attacks oils with vigor should be improved by the administering of another oil. Castor oil seems to contain, some chemical body which is antiseptic in its action. It is well known that oleum ricini very seldom becomes rancid, while the other edible oils spoil in the course of a few days. Other intestinal antiseptics may be of value if the castor oil is poorly borne by the stomach. Hexamethylenamina either alone or in combination with sodium benzoate is an efficient remedy. This may be alternated with sodium salicylate, if the duration of the primary stage is longer than one week.

The diet should be absolutely fat free. This excludes milk, cream, butter, the fat of meat and oils of all kinds. Broths, when free of fat, may be allowed, soups thickened with cornstarch, arrowroot or flour, dry toast, finely divided meat, cooked or raw or skimmed milk.

In the secondary stage, the above named antiseptics may be used alternately. In addition to these, the preparations of arsenic, especially Fowler's solution in small doses will be found of benefit. Occasionally potassium iodide will prove beneficial.

The abstinence from fats and oils must be kept up for several months after improvement has taken place.

In the tertiary stage little can be done. The patient usually needs constant care, either from an attendant or in some institution.

In the treatment of enterocolitis constipation is a very troublesome symptom. So strongly has the importance of free evacuations of the bowels, and the so-called constipation high up and its deleterious influences upon health and life, been impressed upon the minds of the patrons of the medical profession, that were this doctrine true and appli-

cable to all members of the animal kingdom who possess internal digestive systems, the surface of the earth would be left as an habitat for protozoa, bacteria, molds, yeasts and other organisms, who gain nutrition through their external surfaces, and to no others.

Fortunately, constipation is not such a fatal disease as we have been led to believe. It is a difficult matter to decide what is the normal habit of defecation for various individuals. Some people have normally two stools a day, others, and in truth they may be in the majority, have one per diem. Many others, strange as it may seem, get along extremely well with one or two a week until convinced by some friend that they would be in better health if their stools were more copious and frequent and take measures to accomplish this result. In such cases, trouble is not long in developing. Many instances have been reported in which the normal habit was as infrequent as once a month without serious consequences, so far as bodily health and comfort were concerned. When a marked putrefaction is present in the intestinal contents, constipation may become a disagreeable, if not a dangerous symptom. He who attempts to cure sufferers from enterocolitis through the use of cathartics, however, will find that the results of treatment will be uniformly unsatisfactory.

In connection with the subject of constipation, it is found that the contents of the colon are not, in health, very rich in nutritive materials. The colon may be regarded as the reclamation plant of the gastrointestinal canal. Within its cavity, the greater part of the water, mucus, enzymes and hormones are recovered and turned back again into the blood and lymph streams for future use in the digestive process.

When through catharsis, the contents of the small intestine have been moved forward into the colon before the absorption of proteids, carbohydrates and fats has advanced to a proper degree, putrefaction is greatly favored. If this condition is present day after day, in a greater or less length of time, through an overabundance of nutritive material favoring the growth of putrefactive bacteria, a catarrhal condition will most certainly be established.

The stools in enterocolitis often, under analysis, show a bacterial content, living, dead and autolysed, of ninety-nine per cent, according to the investigations of Strasburger and Herter. Consequently it may be assumed that the more copious the stools, the greater the putrefaction. Therefore, a patient who is passing one pound of fecal matter a day is losing 0.99 of a pound of nutrition, plus the amount of carbon, hydrogen and nitrogen that is wasted as gaseous bodies in bacterial metabolism.

The intestinal condition in constipation may be expressed by the following equation:

(1) O+C+I+T.

In which O may represent obstipation; C, a catarrhal process; I, an infection with the bacteria of putrefaction, and T, the toxemia arising therefrom.

It is easy to see that in the presence of certain very toxic members of the families Bacterium and Hyphomycetis, this condition may be dangerous to the patient's welfare and more especially to the integrity of some of the more delicate organs of metabolism and excretion. It is difficult to see, however, how any great harm could be done if the intestinal conditions were so simplified that the equation would read as follows:

(2) O+C.

Or, bearing in mind the fact that the mass of the stool varies with the bacterial activity, what damage could possibly come to the general parenchyma of our patient, even if he should have but one stool a week, if his intestinal condition could be represented by

(3) O.

As a corollary, O may be replaced by D, representing diarrhoea, in equations 1, 2 and 3. In this case we have the condition present in many of our patients, who have contracted intestinal diseases in tropical climates and to whom the intestinal equation O+C+I+T would give a marked degree of comfort, as well as, in comparison, the greatest satisfaction.

The use of cathartic medicines is not to be recommended and their long-continued administration will make all chronic conditions much worse. Even in conditions of intestinal atony without catarrh the administration of such drugs will eventually cause a colitis or an enterocolitis to develop.

Under the teaching of Lane, highly purified mineral oils have come much into vogue for the relief of constipation. The study of feces during such treatment, however, leads to the belief that these oils cause an increase of putrefaction in the intestines.

The various vegetable gums were also tested as intestinal lubricants. Agar agar gave fair results. Finely ground sea moss of the variety chondrus crispus, however, seems to be the best. It has the advantage of being a native product of low cost, is very well borne by the stomach, is soothing to the intestinal mucosa, produces large well formed stools and seems to check rather than encourage putrefaction.

CHAPTER XV

GENERAL CONSIDERATION OF ENTEROCOLITIS

Many of the statements made in the literature of enterocolitis do not bear the test of laboratory investigation. The two most commonly reiterated doctrines, the importance of overeating as a causative factor and the bad effect of consuming certain common articles of food in abundance, have been proven to be without foundation. There are, of course, a great many sufferers from this disease who habitually overeat and who have done so all their lives, but the fact that they have contracted a chronic catarrh of the intestinal tract must be accounted for on other grounds than an overindulgence in the pleasures of the table. In the majority of patients conditions bordering on starvation are more generally found.

Through the teaching of our friends, the vegetarians, the opinion has become very general, that animal foods have a certain poisonous effect upon both mind and body, that their use leads to early decay of the blood vessels and that their consumption at best is but a relic of barbarism. People are all the more ready to accept this belief, as the price of meat products has risen to an alarming extent in the past twenty years and the cost of living may be materially reduced by following a meat-free diet. Moreover, cheating the stomach seems to be a very popular pastime among a certain class of people and routines directed to the relief of abdominal symptoms through starvation treatment are coming more and more into vogue.

Another great cause of intestinal damage is the very prevalent use of foods that have been cooked several weeks or months previous to consumption. This applies not only to meats and vegetables, but to cereals as well. This condition

may be accounted for partly by the well ordered advertising campaigns conducted through the columns of the press by enterprising manufacturers of such products, but in a greater degree through the innate laziness of the average American cook and the widespread ignorance of the art of good cooking. We are rapidly becoming a tin can and carton race, so far as our food supply is concerned. Even in the rural districts, the tin can has become ubiquitous and the number of empty ones found on the premises may be said to vary inversely with the thrift of the family. Truly it is shameful to find tin cans labeled "pork and beans" upon the premises of any self-respecting farmer. On the other hand, in the best hotels in the land the use of canned vegetables is, if not extremely common, the rule. If a vegetable served on these tables is fresh, even when common in the markets at the time, the fact is remarked upon and used as an excuse for an advance in price. One often wonders what may be the age of the canned goods served in the average restaurant.

Meat is often a cause of trouble. Some of it served in public eating houses, through age or maltreatment, is irritating to the intestinal mucosa, to say the least. The methods of the storage of meat, poultry and eggs is often open to criticism. Meat that requires the liberal use of highly aromatic condiments to disguise the odor and taste of advancing putrefaction is well known to be dangerous, yet it is served every day in every city.

Another cause for remark is the filthy condition that exists in the ice boxes, kitchens and serving room, and the uncleanliness of the personnel, not only in public eating houses but in many homes as well. It may seem an exaggeration to state that the cook who prepares our food, the waiter who serves it in so polished a manner, or the scullion who cleans the eating utensils, may communicate sickness, but when one bears in mind the great prevalence of fecal matter upon the hands and person of the average individual, the subject takes a different aspect. It must not be forgotten that the chef may have an intestinal fauna to which he is immune, but which on the other hand may lead to a premature death

or at least months or years of incapacity to one of the guests he might inadvertently infect.

New strains of micro-organisms, introduced through the agency of immigration and travel, no doubt are beginning to have a marked influence upon the intestinal condition of our native population. The intestinal fauna in some of our patients who have lived in the Orient is often remarkable and the effect that the bacteria that these people harbor without inconvenience may have upon the health and vitality of the American born, furnishes a very interesting problem and one deserving of careful study. Possibly, the great decrease in the fecundity of our population may be ascribed to various chronic infections against the attack of which the individual has not acquired an immunity. It is a fact that a conquest is going on through the agency of intestinal putrefactions and of plasmodiasis, instead of by force and arms, that will eventually change the character of the race.

The change from country to urban life, the lack of fresh air and sunshine, the intimate contact with sources of infection that a city life necessitates, the competitions, worries and anxieties of modern life, all must have their effect.

The connection between this catarrh and other general chronic infections such as tuberculosis and malaria is very intimate. So important is this fact that tubercular and malarial infections may be said to be the most common complications of the secondary stage of enterocolitis.

The financial and political loss to the nation resulting from intestinal infections presents one of the most serious problems of enterocolitis. Through its attack the length of life and efficiency of its victims are reduced about twenty-five per cent. It is a greater and more far-reaching problem than the hook worm disease or tuberculosis presents today.

Sufferers from enterocolitis fall an easy prey to habit-forming drugs. Especially liable are they to use habitually the insidious coal tar series or the proprietary articles that contain them. Morphine claims a good many victims, and, on account of the depression of sexual desire that accompanies many types of putrefaction, the aphrodisiac effect of cocaine

is often sought. The persistence of an acetanilid habit is not generally realized. It is so easily obtained in the open market, its use causes so little comment, so many supposedly innocuous proprietary compounds contain it in large proportion, that once formed the habit is hard to break. A vast number of popular beverages and nostrums depending upon caffeine for their stimulating effect are also very popular with these patients and these combined with the ubiquitous cocktail and other alcoholic cordials and tipples add their quota to the damage day by day.

The average American is to-day not only underfed but also underclothed. The thinness of clothing has become a popular fad. As a result the resistance of the body is impaired by overheated homes and offices.

The prevention of this disease presents a problem intimately connected with the prolongation of human life. The return to a simpler life, with lessened worry and anxiety; less time and money spent in the pursuit of pleasures, that must be taken in the end from the family food and clothing; plainer and better food, better cooked; a more evenly balanced diet; the abstinence from irritating foods and condiments; the avoidance of habitual catharsis; and finally more fresh air and sunshine; all suggest themselves as measures that may well be used in the prevention of enterocolitis.